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Emergency Medicine Practice

Evidence-Based Education • Practical Application

CLINICAL CHALLENGES:

- **What are the primary etiologies of right heart failure (RHF)?**
- **Which diagnostic studies should be ordered for patients with suspected RHF?**
- **What are the most effective treatment strategies for RHF, based on underlying etiologies?**

Authors

Daniel S. Brenner, MD, PhD
Assistant Professor of Clinical Emergency Medicine, Department of Emergency Medicine, Indiana University School of Medicine, Indianapolis, IN

Onyedika J. Ilonze, MD
Division of Cardiovascular Medicine, Krannerat Cardiovascular Research Center, Indiana University School of Medicine, Indianapolis, IN

Shelby Beil, MD
Fellow, Indiana University School of Medicine, Department of Pulmonary and Critical Care Medicine, Indianapolis, IN

Kellie Kaneshiro, AMLS
Librarian, Ruth Lilly Medical Library, Indiana University School of Medicine, Indianapolis, IN

Nicholas E. Harrison, MD, MSc
Assistant Professor, Department of Emergency Medicine, Indiana University School of Medicine, Indianapolis, IN

Peer Reviewers

James Morris, MD, MPH, FACEP
Program Director, Emergency Medicine Residency, Texas Tech University Health Sciences Center, Lubbock, TX

Kestrel Reopelle, MD, MEd
Assistant Professor; Assistant Program Director, Residency in Emergency Medicine, Icahn School of Medicine at Mount Sinai, New York, NY

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Emergency Department Management of Patients With Right Heart Failure

Abstract

Right heart failure (RHF) can result from many cardiac, pulmonary, and systemic pathologies. Common causes of RHF include pulmonary embolism, left heart failure, congenital heart disease, chronic lung disease, acute myocardial infarction, infiltrative disease, infectious disease, and valvular abnormalities. Acute and chronic RHF confer a high risk for morbidity and mortality in the acute care setting, and interventions commonly used in emergency care can prompt acute decompensation if the RHF is not recognized. The severity of presentation may range from compensated clinically silent cardiovascular dysfunction to venous congestion, multiorgan failure, and circulatory collapse. This review describes the pathophysiology of right heart failure and offers an evidence-based approach to the diagnosis, management, and disposition of both acute and chronic RHF.

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Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this activity, you should be able to: (1) identify areas in practice that require modification to be consistent with current evidence in order to improve competence and performance; (2) develop strategies to accurately diagnose and treat both common and critical ED presentations; and (3) demonstrate informed medical decision-making based on the strongest clinical evidence.

CME Objectives: Upon completion of this activity, you should be able to: (1) outline the etiologies of right heart failure (RHF)/right ventricular dysfunction (RVD)/pulmonary hypertension (PH); (2) describe the recommended diagnostic studies for RHF; (3) describe treatment strategies for RHF, based on etiology; and (4) explain the importance of risk stratification of patients with RHF/RVD/PH for disposition.

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Direct all inquiries to:
Phone: 678-366-7933
Fax: 770-500-1316
5600 Spalding Drive, Unit 921697
Norcross, GA 30010-1697
E-mail: **ebm@ebmedicine.net**
Website: **www.ebmedicine.net**

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Case Presentations

CASE 1

A 40-year-old woman presents with dyspnea, tachycardia, and hemoptysis…

- Her vital signs are: temperature, 36.9°C; heart rate, 125 beats/min; blood pressure, 120/65 mm Hg; respiratory rate, 24 breaths/min; and oxygen saturation, 94%.
- She takes estrogen-containing medications, and reports that she recently had a cross-continental airplane flight.
- As you place initial orders, you consider whether transthoracic echocardiography would aid in her evaluation and management…

CASE 2

A 64-year-old man with a history of heart failure reports severe fatigue and dyspnea…

- His vital signs are: temperature, 36.5°C; heart rate, 115 beats/min; blood pressure, 150/90 mm Hg; and respiratory rate, 26 breaths/min. In the ED, his resting oxygen saturation is 93%.
- He states that he is compliant with his diuretic therapy, but he has developed worsened lower extremity edema.
- What aspects of cardiac function most acutely correspond to his symptoms and risk for adverse events?

CASE 3

A 52-year-old woman with COVID-19 and severe respiratory distress develops hemodynamic instability immediately after you intubate her…

- Her vital signs are: temperature, 37.5°C; heart rate, 130 beats/min; blood pressure, 75/42 mm Hg; and respiratory rate, 22 breaths/min.
- What are the likely causes of her shock, and what should you do to stabilize her?

Introduction

Right heart failure (RHF) is the common endpoint of numerous cardiac, pulmonary, and systemic pathologies, and can present a high risk for patient morbidity and mortality in the acute care setting. Presentation severity runs a spectrum from compensated, clinically silent cardiovascular dysfunction to venous congestion, multiorgan failure, and circulatory collapse.[1]

While RHF is common among emergency department (ED) patients, diagnosing and managing RHF is challenging.[1] Specific hurdles include etiologic heterogeneity, nonspecific clinical presentations, comorbid complexity, potentially harmful effects of common ED treatments, and a limited armamentarium of diagnostic and therapeutic tools available to ED clinicians. The presence of RHF can dramatically alter the risk-benefit profile of management and disposition paradigms for common ED conditions, making recognition of RHF vitally important. This issue of *Emergency Medicine Practice* presents an evidence-based approach to the diagnosis, management, and disposition of patients with RHF.

Selected Abbreviations

ARDS	Acute respiratory distress syndrome
COPD	Chronic obstructive pulmonary disease
CT	Computed tomography
CTEPH	Chronic thromboembolic pulmonary hypertension
CTPA	CT pulmonary angiogram
ECG	Electrocardiogram
ECMO	Extracorporeal membrane oxygenation
HFrEF	Heart failure with reduced ejection fraction
HFpEF	Heart failure with preserved ejection fraction
LVAD	Left ventricular assist device
MI	Myocardial infarction
NIPPV	Noninvasive positive pressure ventilation
PASP	Pulmonary artery systolic pressure
PEEP	Positive end-expiratory pressure
PPV	Positive pressure ventilation
RHF	Right heart failure
RVD	Right ventricular dysfunction
RVMI	Right ventricular myocardial infarction
RVSP	Right ventricular systolic pressure
RV/LV	Ratio of size of right ventricle to left ventricle
STE	Speckle-tracking echocardiography
TAPSE	Tricuspid annular plane systolic excursion
TTE	Transthoracic echocardiography

Critical Appraisal of the Literature

A keyword and medical subject heading (MeSH) search was performed of 8 databases: MEDLINE®, American College of Physicians Journal Club, the Center for Clinical and Translational Research, the Cochrane Database of Systematic Reviews, the Cochrane Methodology Register Database, the Health Technology Assessment Database, the National Health Service Economic Evaluation Database, and the Database of Abstracts of Reviews and Effects. Fifty-three MeSH terms comprised the search from

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1990 to 2022. Human original research, systematic reviews, and professional society guidelines were considered if involving acute and acute-on-chronic RHF and/or pulmonary hypertension. Chronic RHF articles were considered if they impacted ED care. The search retrieved 4865 articles that were adjudicated by 2 reviewers for relevance to the topic at 2 stages: title and abstract screening (n=1004), and full text review (n=283). Other topic reviews on RHF in ED or critical care (n=26) were not cited directly, instead having their reference lists screened for research not captured by the primary search (n=1). When background information for niche subjects was necessary to provide context for articles in the primary search, articles outside the search (n=7) were included. Evidence was predominantly observational and of overall low quality. The few randomized trials present were small and designed to test low-impact outcomes rather than patient-centered endpoints. Final citations were chosen to favor original research with large sample sizes, randomized trials, questions of clinical importance to emergency medicine clinicians, areas in which several small/low-quality studies agreed on principal conclusions, systematic reviews, and practice guidelines.

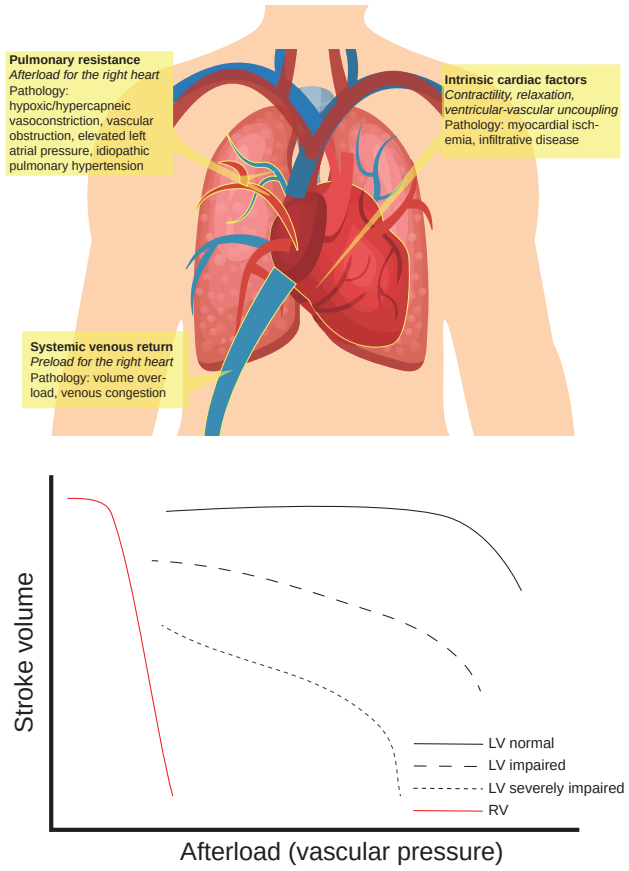
Etiology and Pathophysiology

Right heart function is dictated by 3 basic physiologic domains: (1) resistance to right ventricular ejection (afterload) via pulmonary artery pressure; (2) right ventricular systolic and diastolic pump function; and (3) right heart filling (preload) via systemic venous return. **(See Figure 1.)** The pathophysiology of RHF can be conceptualized by the affected domain: increased afterload (pulmonary hypertension), reduced right ventricular contractility or compliance, and/or excess preload (volume excess, elevated central venous pressure).[1] At the "tipping point" from compensated to decompensated RHF, typically all 3 domains are impaired, due to self-propagating feedback loops wherein dysfunction of any single domain precipitates worsening of the other domains. **(See Figure 2, page 5.)**

Pulmonary hypertension is associated with nearly all etiologies of RHF.[1] The thin-walled right ventricle is profoundly afterload-sensitive compared to the left ventricle, since mean pulmonary artery pressure is normally minimal (<25 mm Hg) compared to aortic pressures. Therefore, only small increases in afterload are necessary to cause acute or chronic RHF. Pulmonary hypertension can develop as a progressive chronic disease and/or by rapid severe decompensation and is categorized by 5 etiological "groups." **(See Table 1, page 6.)** Acute pulmonary vascular obstruction (eg, pulmonary embolism) and hypoxemic vasoconstriction of pulmonary vessels are 2 of the fastest and most dramatic mechanisms behind rapid elevation of pulmonary artery pressure and concomitant right ventricular failure.[1]

Right ventricular dysfunction is most often related to increased afterload or preload but can also occur independently. One essential concept is ventricular interdependence. Right ventricular and left ventricular contractility and relaxation are coupled because they share the interventricular septum and a fixed space (the pericardium) within which to accommodate pressure-related chamber dilation. As a result, a proportion of right ventricle systolic function comes from left ventricle contraction under normal conditions, and left ventricular dysfunction conveys right ventricular dysfunction (RVD) through the septal interactions. This also impacts right ventricular function through pressure differentials in the context of a shared cardiac output. Disproportionate elevations in right ventricle versus left ventricle pressures, or right ventricle versus pulmonary artery pressures, cause

Figure 1. Pathophysiology of Right Heart Failure



The pathophysiology of right heart failure can be conceptualized as the joint dysfunction of 3 domains: (1) pulmonary artery pressure (afterload); (2) right ventricular dysfunction (impaired contractility, relaxation, and/or ventricular-vascular coupling); and (3) elevated right atrial pressure (preload). Right ventricular stroke volume (as seen in the red line on the graph) declines rapidly with increased filling pressure.
Abbreviations: LV, left ventricle; RV, right ventricle.
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disorganized contractility and reduced filling efficiency.[2] This can occur from uncoupling of the ideal ventricular-vascular relationship: pulmonary artery pressure and systemic vascular resistance must (by Starling's Law) change in an exact inverse magnitude to right ventricular and left ventricular end diastolic volume (respectively) to maintain constant cardiac output. Finally, right ventricular ischemia impairs right ventricular contractility. Most commonly, right ventricular ischemia is a result of demand/perfusion mismatch and the effect of elevated transmural right ventricular pressures, but it can be secondary to acute coronary occlusion as well.[1]

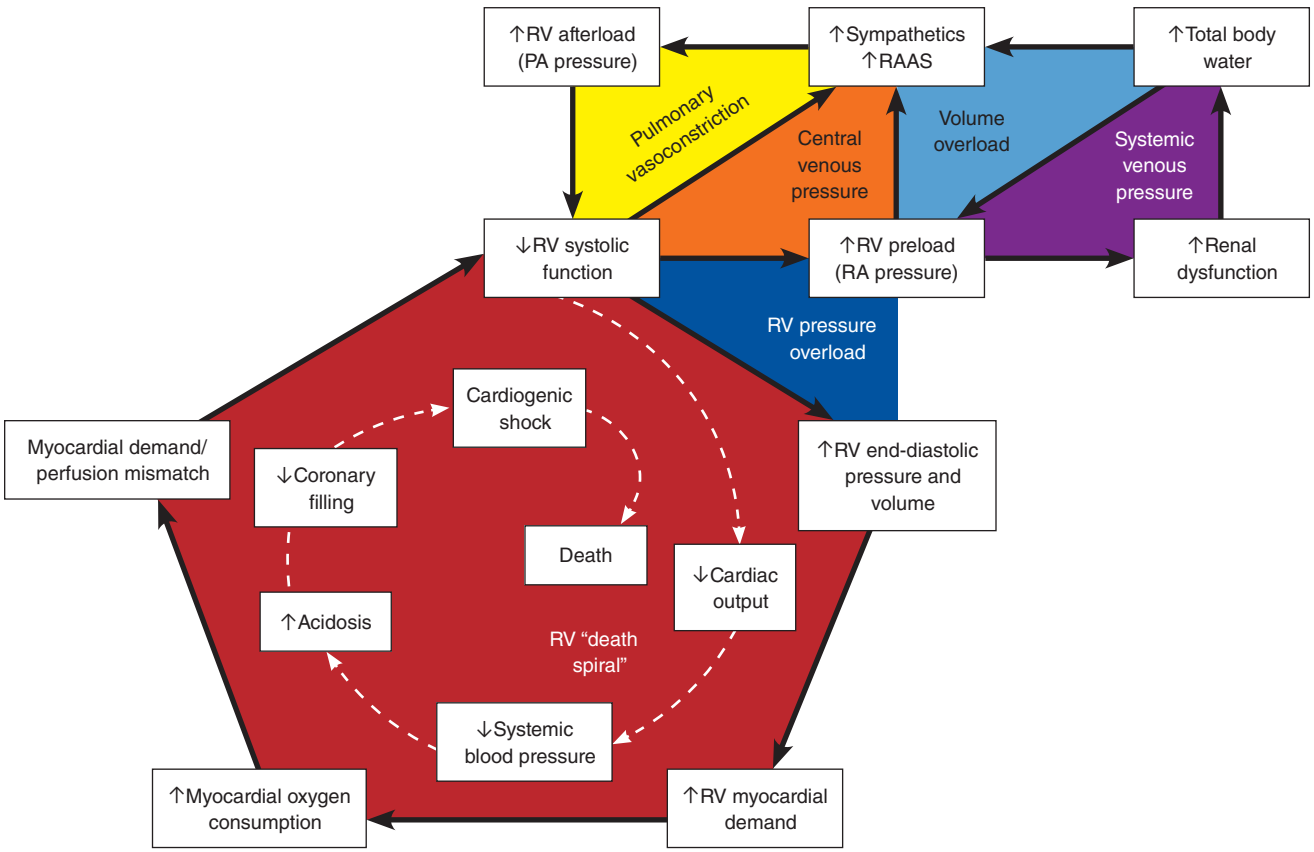
Lastly, venous congestion is both a cause and effect of elevated right ventricular pressure and, without rapid intervention, can become a self-propagating cycle. **(See Figure 2.)** Elevated right ventricular pressure impairs contractility and eventually causes right ventricular ischemia.[1] When superimposed on increased afterload and/or intrinsic RVD, excessive preload contributes to the self-perpetuating cycle of hypoxia, acidosis, and ischemia that is sometimes referred to as the "right ventricular death spiral." Venous congestion is also the primary mechanism of systemic effects of RHF, including cardiohepatic and cardiorenal syndromes.[1]

Differential Diagnosis

The differential diagnosis for RHF is broad, due to its nonspecific symptoms: fatigue, exercise intolerance, shortness of breath, chest pain, lower extremity edema, syncope, early satiety, and arrythmia.[3,4] A thorough history and physical examination are critical in differentiating RHF from other etiologies. Many patients with new-onset pulmonary hypertension or acute RHF have identifiable predisposing risk factors.

Diagnosis of medical conditions associated with RHF (acute pulmonary embolism, sepsis, heart failure) may lead to identification of secondary RHF. In other

Figure 2. Maladaptive Feedback Loops in Right Heart Failure



In RHF, increased afterload, excessive preload, and right ventricle systolic dysfunction form self-propagating feedback loops with one another. If RHF progresses to overtly depressed cardiac output, an additional feedback loop of worsening ischemia, acidosis, and cardiogenic shock may develop. This so-called "right ventricular death spiral" (red pentagon) is usually intractable, with rapid deterioration to hemodynamic collapse and death. Thus, clinical care of RHF is most successful when focusing on aggressive relief of the early mechanisms, with a goal of stabilizing right heart function prior to the development of shock.

Abbreviations: PA, pulmonary artery; RA, right atrium/atrial; RAAS, renin-angiotensin-aldosterone system; RHF, right heart failure; RV, right ventricular.

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cases, a patient may be diagnosed with undifferentiated RHF first, without a clear etiology. The latter is particularly challenging because possible causes of RHF are vast, and more than one acute or chronic etiology may contribute.[5] **(See Table 2.)**

Causes of Acute Right Heart Failure

Acute Pulmonary Embolism

Pulmonary embolism is the most common cause of acute RHF. Lower extremity deep vein thrombi, and less commonly other products (amniotic fluid, air, or septic emboli), embolize to the pulmonary arterial tree and obstruct circulatory flow. Obstruction rapidly increases right ventricular afterload, often proportionally to total clot burden and clot location.[6,7] The increased afterload is sufficient to cause RVD and acute RHF in 50% to 60% of pulmonary emboli, and overt obstructive shock in 5% to 10%.[8] RHF is the primary source of morbidity and mortality in pulmonary embolism.[6,7]

Sepsis

In a prospective study of 252 septic ED patients, 34% of patients with sepsis or septic shock exhibited RVD, occurring with concomitant left ventricular systolic dysfunction in 71% of those patients.[9] Pathophysiology is poorly understood and may represent a combination of "unmasked" underlying cardiac disease, de novo septic cardiomyopathy due to inflammatory and neurohormonal cascades, secondary effects of multiorgan dysfunction (eg, renal and hepatic failure, acute respiratory distress syndrome [ARDS]), and iatrogenic effects of common interventions (aggressive intravenous [IV] volume loading, positive pressure ventilation (PPV) and acute cor pulmonale).[1] Signs and symptoms of RHF overlap with multiorgan dysfunction in sepsis, and patients with RVD have dramatically poorer outcomes than those without.[9]

Right Ventricular Myocardial Infarction

RHF due to acute right ventricular myocardial infarction (RVMI) should be suspected in patients with hypotension, jugular venous distension, clear lung fields, and symptoms consistent with acute myocardial ischemia. Isolated RVMI is uncommon (3% of all myocardial infarctions [MIs]), but right ventricular ischemia complicated up to half of all inferior MIs in a retrospective study of 232 patients.[10] Heart failure is common after RVMI and can worsen its clinical course.[2] Moreover, as many as one-third of anterior acute MIs may result in RVD.[11] RVMI disproportionately accounts for acute MIs complicated by cardiogenic shock and a high burden of arrythmias.[1] Causes of acute RVMI include acute type 1 MI, spontaneous coronary dissection, and dislodgement of

Table 2. Etiologies of Acute Right Heart Failure

Acute Right Heart Failure

- Pulmonary embolism
- Septic cardiomyopathy
- Right ventricular myocardial infarction
- Acute valvular insufficiency
- Acute cor pulmonale (due to ARDS, COVID-19, positive pressure ventilation, pneumonia)
- Post cardiac surgery
- Post left ventricular assist device placement

Acute-on-Chronic Heart Failure

See Table 1 for a more complete list of chronic RHF comorbidities (all can experience acute exacerbation)

The most common causes include:

- Biventricular acute heart failure (group 2 PH)
- COPD and other chronic hypoxic disease (group 3 PH)

Abbreviations: ARDS, acute respiratory distress syndrome; COPD, chronic obstructive pulmonary disease; PH, pulmonary hypertension; RHF, right heart failure.

Table 1. Classification of Pulmonary Hypertension and Comorbidities Associated With Right Heart Failure

Classification	Etiology/Mechanism	Associated Comorbidities
Group 1	Idiopathic elevated pulmonary arterial pressure	Pulmonary arterial hypertension
Group 2	Elevated left arterial pressure transmitted to pulmonary artery, interventricular dependence	Left heart failure
Group 3	Chronic lung disease and/or hypoxia → vasoconstriction and/or destruction of pulmonary capillary beds	COPD, pulmonary fibrosis, obstructive sleep apnea, obesity hypoventilation syndrome, cystic fibrosis, infiltrative lung disease (sarcoidosis, amyloidosis), pneumoconiosis, bronchiolitis obliterans
Group 4	Pulmonary arterial obstruction	Thromboembolic disease, systemic lupus erythematosus with hypercoagulable features, CTEPH
Group 5	Various mechanisms/multifactorial	Sickle cell disease, chronic kidney disease, chronic hemolytic disease
Primary cardiac	Cardiac structural or infiltrative abnormalities	Cardiac congenital anomalies, cardiac surgery, LVAD, amyloidosis, systemic rheumatic diseases, Chagas disease, HIV

Abbreviations: COPD, chronic obstructive pulmonary disease; CTEPH, chronic thromboembolic pulmonary hypertension; LVAD, left ventricular assist device.

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an inserted cardiac device. In a retrospective cohort of 939 patients, occlusions proximal to the right ventricle marginal artery were associated with larger infarction size and higher rates of cardiogenic shock and mortality.[12]

Positive Pressure Ventilation and Acute Respiratory Distress Syndrome

Invasive PPV increases intrathoracic pressure, which may compress the pulmonary vasculature and increase right ventricular afterload. Elevated intrathoracic pressure also reduces central venous return, causing decreased right ventricular preload. In patients with impaired right ventricular function, depressed right ventricular preload can improve hemodynamics for those who are volume/pressure overloaded, or it can precipitate hemodynamic collapse in patients who are euvolemic or hypovolemic. **(See Figure 3.)**

Acute Respiratory Distress Syndrome

In a retrospective study of 583 patients, acute RHF was a complication of ARDS in 25% to 50% of cases.[13] The effects of PPV are compounded by hypoxia and permissive hypercapnia contributing to pulmonary vasoconstriction. The high positive end-expiratory pressure (PEEP) strategy commonly used in ARDS leads to increased right ventricular afterload and decreased right ventricular preload, worsening right heart function. A single-center retrospective review of 352 patients with ARDS showed mortality and the rate of acute cor pulmonale are directly correlated with increasing plateau pressure.[14] A recent meta-analysis including 1861 patients showed that patients with ARDS complicated by RVD have significantly higher mortality (odds ratio [OR], 1.45).[15]

COVID-19

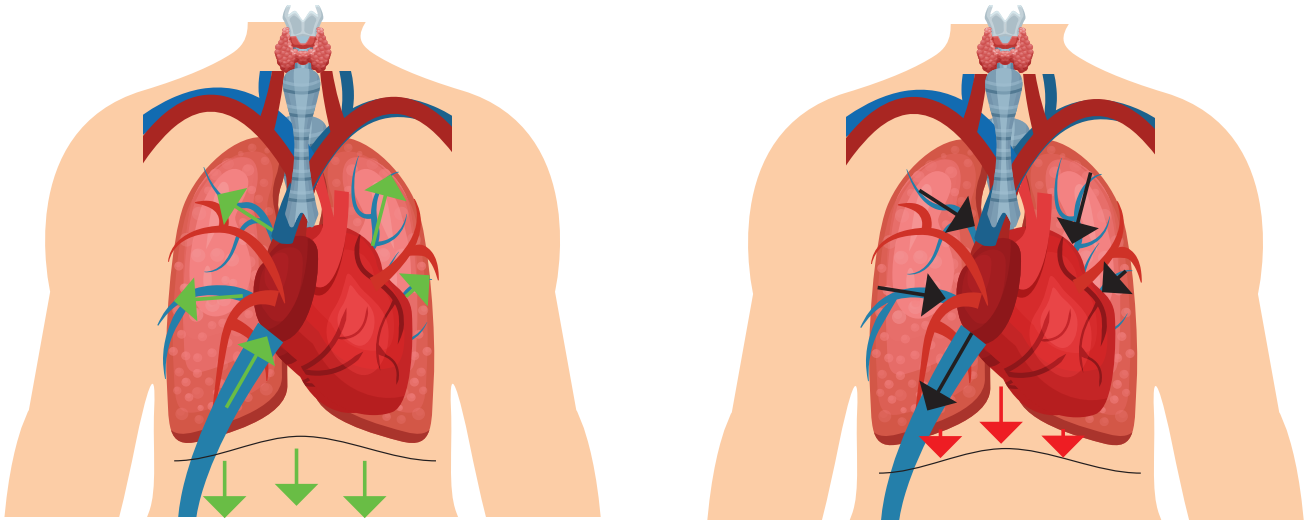
Severe COVID-19 infection is associated with acute RHF (via physiology similar to ARDS and PPV), along with an increased incidence of thromboembolic disease. A prospective cross-sectional study assessed cardiac function in patients with severe COVID-19 within 24 hours of intubation and demonstrated that 39% had right ventricular dilation, and 14% developed acute cor pulmonale.[16] Right ventricular dysfunction is a poor prognostic factor in severe COVID-19, with mortality of 61.5% in patients with acute RHF compared to 12.8% without.[17]

Causes of Chronic Right Heart Failure

Chronic Thromboembolic Pulmonary Hypertension

Chronic thromboembolic pulmonary hypertension (CTEPH) is a chronic disease wherein persistent pulmonary vascular obstruction leads to chronic pulmonary hypertension and RHF. It is thought that chronic residual thrombus may undergo fibrous organization, which renders it less susceptible to treatment by thrombolysis.[18] An estimated 4% to 12% of patients in a retrospective cohort of 358 patients with acute pulmonary embolism developed CTEPH.[18] A substantial proportion of CTEPH cases occur in the absence

Figure 3. Hemodynamic Effects on the Right Heart and Pulmonary Vasculature


Negative pressure inspiration
With normal breathing, negative intrathoracic pressure pulls blood in from the inferior vena cava (increasing RV preload), and pulls blood in from the ventricles (reducing RV afterload)
Positive pressure inspiration
With positive pressure ventilation, positive intrathoracic pressure pushes blood away in inferior vena cava (reducing RV preload), and pushes blood away from the lungs (increasing RV afterload)

Hemodynamic effects on the right heart and pulmonary vasculature with negative-pressure ventilation (regular breathing) compared with positive pressure ventilation.
Abbreviation: RV, right ventricular.
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of known prior pulmonary embolism history.[4,18,19] In multiple retrospective studies (n=358, n=219, n=50) progression to CTEPH was more common when prolonged RVD was present on transthoracic echocardiography (TTE) after treatment for acute pulmonary embolism (OR, 7.14; 95% confidence interval [CI], 2.15-23.78).[18-20]

The criteria for CTEPH are based on elevated persistent right ventricular pressures, and right-heart catheterization is typically necessary for diagnosis. Computed tomographic pulmonary angiography (CTPA) is insufficient to exclude the diagnosis.[4] Diagnostic criteria also include persistent pulmonary hypertension for at least 3 months on appropriate anticoagulation therapy to differentiate CTEPH from "subacute" pulmonary embolism[7] and exclusion of pulmonary arterial hypertension.[4] Patients with unexplained pulmonary hypertension and/or chronic RHF with or without a history of acute pulmonary embolism therefore need urgent referral to a pulmonary hypertension specialist for diagnosis.[4] CTEPH is one of the few causes of chronic pulmonary hypertension for which etiology-specific therapy (pulmonary endarterectomy) can be effective at reversing the disease.[4]

Heart Failure

Patients with heart failure and reduced or preserved ejection fraction (HFrEF and HFpEF, respectively) can have RHF. A meta-analysis of 81 studies suggested that patients with HFrEF exhibit RVD in 48% of cases, while the incidence of RVD or pulmonary hypertension in HFpEF is estimated at 33% to 68% of patients.[1,21] Right ventricular dysfunction confers a 2.4-fold increased risk for mortality, urgent transplantation, or urgent left ventricular assist device (LVAD) implantation at 90 days in HFrEF, and a 7-fold higher mortality in HFpEF.[1]

In a prospective observational trial of 84 patients, at the time of ED presentation for acute heart failure, 53% of patients had RVD and/or pulmonary hypertension, yet these were unrecognized 96% of the time.[22] Thus, around half of "left-sided" heart failure is biventricular, and failure to recognize right-heart involvement is common. A variety of mechanisms underlie this association: increased right ventricular afterload from post–capillary pulmonary hypertension; volume overload, causing demand-perfusion mismatch in the pressure-sensitive right heart; interventricular dependence; and shared right ventricle/left ventricle cardiac output in the presence of ventricular-vascular uncoupling.[1] In this trial, after adjusting for left ventricular function and other confounders, RVD at ED arrival was associated with longer length of stay (5.51 vs 3.67 hospital days) and 4-fold greater odds of 30-day adverse outcomes.[22] Aggressive treatment is essential for these patients, as significant improvement in right ventricular systolic function was observed just 24 hours after the first ED treatment for acute heart failure (46% to 38%, adjusted OR, 0.08; 95% CI, 0.02-0.48).[21,22]

Congenital Heart Disease

RHF is a component of many congenital cardiac anomalies due to right-sided valvular lesions, intracardiac shunts, and/or the presence of a systemic right ventricle.[1] **(See Table 3.)** Furthermore, therapy for some forms of congenital heart disease requires connection of the right ventricle to the systemic circulation, leading to right ventricular pressure overload and eventually RHF. The most important aspect of

Table 3. Congenital Cardiac Conditions Associated with Right Heart Failure

Condition	Mechanism of Right Heart Failure	Notes
Atrial septal defect	Left-to-right shunt, Eisenmenger physiology	Often asymptomatic in childhood.
Ebstein anomaly	Atrialization of the right ventricle due to adherence of tricuspid leaflet to ventricular wall	One of the few causes of primary tricuspid regurgitation.
Pulmonary stenosis	Right ventricular outflow obstruction	Component of 10% of CHD. May be isolated or part of a complex CHD syndrome.
Ventricular septal defect	Left-to-right shunt	May occur in isolation, or as component of a complex syndrome.
Hypoplastic left heart syndrome	Single right ventricle physiology	After repair (Fontan procedure), a single systemic ventricle is formed. RHF is common by the third or fourth decade of life after the Fontan procedure.
Levo-transposition of the great arteries	Connection of the LV to the pulmonary artery, and the RV to the aorta	Requires additional lesions (VSD, patent ductus arteriosus) for survival. Repair can involve a systemic RV, and therefore RHF. RHF is particularly common in levo-transposition of the great arteries. There is 50% RHF prevalence by middle age.
Tetralogy of Fallot	VSD, overriding aorta, pulmonary stenosis, right ventricular hypertrophy	Severe pulmonary regurgitation is the most common residual abnormality.

Abbreviations: CHD, congenital heart disease; LV, left ventricle; RHF, right heart failure; RV, right ventricle; VSD, ventricular septal defect.

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caring for patients with congenital heart disease is understanding the patient-specific anatomy and physiological adaptations.

RHF is a common late complication of congenital heart disease, occurring decades after initial repair in 5% to 10% of patients.[4] Among congenital heart disease patients hospitalized for heart failure as adults, two-thirds show signs of RHF.[23] Mortality in this population may, nevertheless, be similar to other patients hospitalized for acute heart failure.[23] Acute evaluation with TTE can be helpful (even though the distorted anatomy may be challenging to interpret) because it can yield information regarding volume status and ventricular function. Detailed descriptions of specific forms of congenital heart disease and their ED management are beyond the scope of this review, but may be found in other resources.[24]

Left Ventricular Assist Devices

Right ventricular failure is one of the most common complications post-LVAD implantation. LVAD implantation improves contractility and forward flow from the left ventricle, which decreases right ventricular afterload. However, increasing cardiac output also increases systemic venous return, which may overload a weakened right ventricle. In a single-center retrospective study of 293 patients, 11% of patients were found to have late RHF after LVAD implantation, which was associated with a 15% absolute reduction in survival.[25]

Lung Diseases and Group 3 Pulmonary Hypertension

RHF due to acute or chronic lung disease, known as *cor pulmonale*, occurs secondary to pulmonary vascular constriction, chronic hypoxia/hypercapnia, and/or loss of pulmonary vascular beds. Ultimately, these factors lead to increased right ventricular afterload and subsequent isolated right ventricular failure. A wide array of obstructive and restrictive lung diseases can lead to this pathophysiology. **(See Table 1, page 6.)** Many etiologies present with acute-on-chronic decompensation, while others can complicate unrelated ED presentations as chronic risk factors.

Pulmonary Arterial Hypertension

Pulmonary arterial hypertension is an idiopathic increase in pulmonary artery tone and resistance. The most common complication of these changes is RHF.[1] Pulmonary arterial hypertension is the only chronic cause of pulmonary hypertension for which chronic pulmonary vasodilator therapy is universally beneficial as a disease-modifying therapy, and discontinuation of these home medications can rapidly precipitate acute-on-chronic RHF.

Prehospital Care

Given the difficulty in diagnosing RHF without TTE, specific, directed care for RHF in the prehospital setting may be challenging. For patients at risk for RHF based on history, prehospital providers should avoid interventions that could worsen RVD and precipitate cardiac arrest, including intubation, phenylephrine, and large IV fluid boluses.[4]

Emergency Department Evaluation

History

The first step in ED evaluation for RHF is a thorough history and chart review, with emphasis on risk factors that could predispose the patient to chronic or acute-on-chronic RHF. **(See Table 1, page 6.)** Next, clinicians should identify whether any of a patient's acute conditions diagnosed during standard ED workup are associated with acute or acute-on-chronic RHF. **(See Table 4.)** Acute RHF most commonly occurs in the

Table 4. Common Emergency Department Diagnoses Associated With Acute Right Heart Failure

Emergency Department Diagnosis	Mechanism(s) of Acute RHF
Acute pulmonary embolus	Pulmonary vascular obstruction, hypoxemic vasoconstriction, acidosis, ischemia
Acute heart failure	Interventricular dependence, elevated left atrial pressure, hypoxemic vasoconstriction, venous congestion[22]
Sepsis	Septic cardiomyopathy, multiorgan dysfunction, iatrogenic volume overload, acidosis
Acute hypoxemic respiratory failure (eg, pneumonia, ARDS, COVID-19, acute exacerbation of chronic lung disease, pulmonary edema)	Hypoxemic vasoconstriction
Acute myocardial infarction	Loss of myocardium
Volume overload	Renal disease, transfusion-associated cardiopulmonary overload
Acidosis	Myocardial impairment and right ventricle-pulmonary artery uncoupling
Positive pressure ventilation	Increased right ventricular afterload
Left ventricular assist device	Right ventricular volume overload and depressed contractility
Myocarditis	Depressed right ventricle contractility

Abbreviations: ARDS, acute respiratory distress syndrome; RHF, right heart failure.

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presence of another critical condition, and recognition of the primary condition will naturally occur first.

Historical symptoms consistent with RHF are largely the same as for isolated left-sided heart failure: dyspnea, chest pain, fatigue, exercise intolerance, orthopnea, paroxysmal nocturnal dyspnea, and edema. Edema is often the most prominent factor in chronic RHF, but may be minimal or absent in acute RHF.[1] Anorexia, abdominal bloating, cachexia, and early satiety occur in RHF due to splanchnic congestion, but are also more common with chronicity. Syncope in a patient with confirmed acute pulmonary embolism is relatively specific for RHF, though it lacked sensitivity in a prospective trial of 335 patients.[26] Unfortunately, no symptoms are highly sensitive or specific for RHF.[1,27]

A new diagnosis of RHF/pulmonary hypertension should also be considered when a patient has dyspnea or fatigue that cannot be attributed to comorbidities. In a prospective cohort of 83 ED patients, 20% to 30% of those with negative CTPA performed for shortness of breath had undiagnosed isolated RHF.[28]

Physical Examination

No physical examination signs are sensitive or specific for RHF. The findings that clinicians associate with left-sided heart failure are essentially the same as those in RHF, including jugular venous distention, hepatomegaly, peripheral edema, and abdominal distention.[1] Society guidelines suggest that a lack of pulmonary edema with other signs of heart failure can suggest isolated right ventricle failure, though this pattern has not been examined in the literature and is thought to lack both sensitivity and specificity, since nearly half of heart failure is biventricular. Unfortunately, auscultation lacks significant accuracy for diagnosing pulmonary edema.[1,22]

Diagnostic Studies

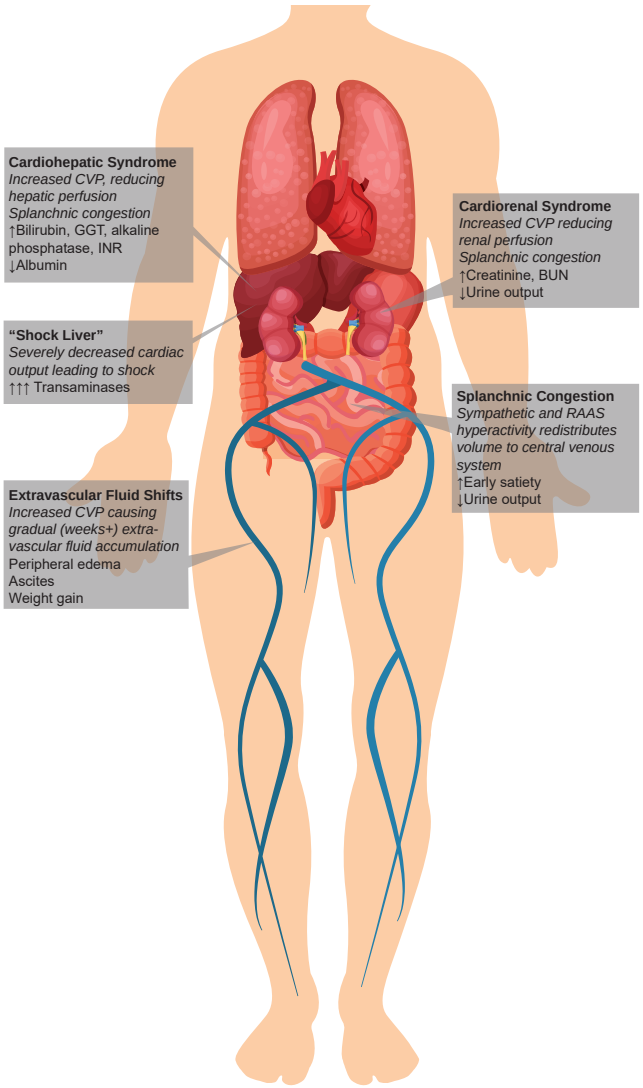
Biomarkers

Laboratory studies are useful in screening for RHF, but they lack specificity for confirming the diagnosis. Brain natriuretic peptide (BNP), N-terminal pro-brain natriuretic peptide (NT-pro-BNP), and troponin are secreted by the myocardium in response to increased ventricular pressure and volume. Thus, as with history and physical examination findings, biomarker elevations will be present in both right and left heart failure.

Biomarkers of renal function can help diagnose cardiorenal syndrome, which is characterized clinically by decreased urine output, fluid retention, and increased diuretic requirements. Cardiorenal syndrome is typically a result of increased central venous pressure, in which elevated renal vein pressure impairs renal perfusion, and therefore is commonly a result of RHF.[1] **(See Figure 4.)**

Laboratory abnormalities include increased blood urea nitrogen and creatinine.[1] If the renal insufficiency is erroneously attributed to prerenal causes rather than as a sequela of venous congestion, delays in initiating diuresis or reduction of loop diuretic therapy can compound worsening renal function.

Figure 4. Extrapulmonary Manifestations of Right Heart Failure



Cardiohepatic syndrome (often referred to as "congestive hepatopathy") is another consequence of right heart failure. Common laboratory abnormalities include markers of cholestasis including elevated bilirubin, gamma-glutamyl transpeptidase, and alkaline phosphatase, as well as markers of altered synthetic function manifested through hypoalbuminemia and prolonged prothrombin time. Substantially elevated transaminases are more commonly related to "shock liver" and should be addressed through treatment of shock rather than aggressive diuresis.[5]

Abbreviations: BUN, blood urea nitrogen; CVP, central venous pressure; INR, international normalized ratio; GGT, gamma-glutamyl transferase; RAAS, renin-angiotensin-aldosterone system.

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Electrocardiogram

A 12-lead electrocardiogram (ECG) is key to prompt a diagnosis of RVMI, and may aid in screening for RHF. ECG signs of both are summarized in **Table 5**. Patients with evidence of inferior occlusive MI should be screened for RVMI with a right-sided ECG; **Figure 5** demonstrates an RVMI on a right-sided ECG.

Table 5. Electrocardiographic Signs Associated With Right Heart Failure

Sign	Significance
• Prominent inferior (>2.5 mm) or V_1, V_2 (>1.5 mm) P waves	Right atrium enlargement
• Dominant V_1 R wave (>7 mm or R/S ratio >1) • Right axis deviation (≥110°) • Dominant S in V_5/V_6 (>7 mm or R/S ratio <1)	Right ventricle hypertrophy
• Right bundle branch block	Right ventricle stretch, hypertrophy, pulmonary hypertension, ischemia, etc
• ST depression/T inversion in V_1-V_4 and/or inferior leads	Right ventricular strain pattern
• Dominant inferior S waves (S_1/Q_3/T_3 pattern)	Support right ventricular hypertrophy
• ST elevation ≥1 mm in V_4R alone **or** • ≥0.5 in multiple leads between V_4R and V_1	RVMI
• Inferior and anterior STEMI	May be associated with RVMI; consider right-side electrocardiogram

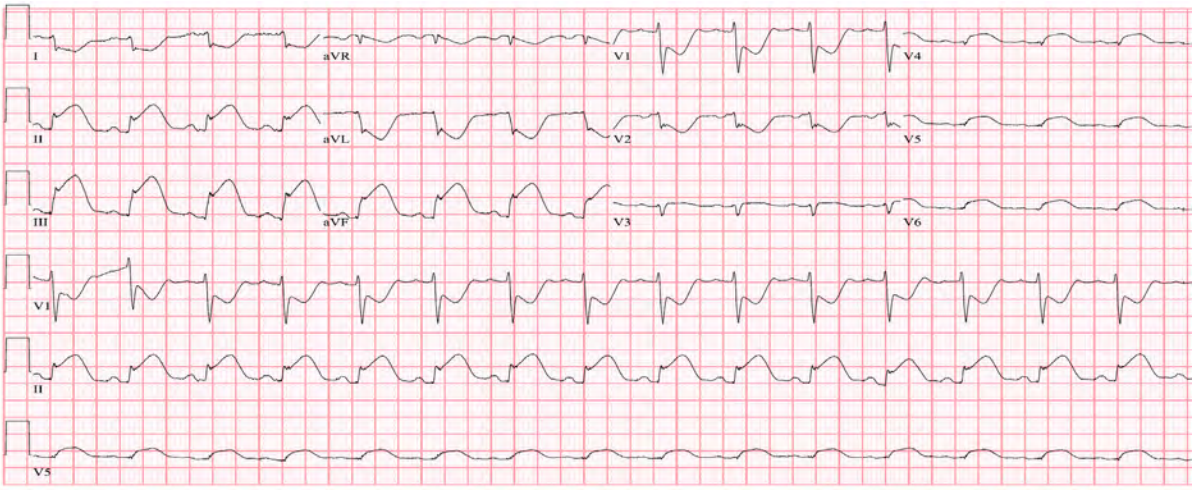
Abbreviations: RVMI, right ventricular myocardial infarction; STEMI, ST-segment elevation myocardial infarction.

ECG is nonspecific and insensitive for RHF diagnosis. However, combination of many signs may additively enhance diagnostic performance. In a prospective study of 103 patients with pulmonary embolism, an "ECG score" of 17 different signs achieved 0.82 area under the receiver operating characteristic curve for TTE evidence of RVD, although this scoring system has not been externally validated.[29]

Transthoracic Echocardiography

Although TTE is the primary diagnostic modality for confirming the presence or absence of RHF, signs of RHF on TTE are nonspecific for underlying etiologies of RHF.[5,31] TTE is rapid, noninvasive, and can be performed at the bedside. Although no single TTE parameter defines RHF and multiple parameters may be interpreted in concert, commonly used measures—including the ratio of the sizes of the right ventricle to the left ventricle (RV/LV), and tricuspid annular plane systolic excursion (TAPSE)—can provide reasonable global assessments of RVD. Novice emergency clinician sonographers show strong correlation in acquisition and interpretation of these 2 measures when compared to formal echocardiography interpreted by cardiologists, with kappa 0.68 (95% CI, 0.48-0.88) for right ventricular dilation and 0.94 (95% CI, 0.87-0.98) for TAPSE.[22,30-34] A normal RV/LV ratio should be <0.8, with larger ratios suggesting right ventricular pressure overload. The RV/LV ratio is most accurately measured in the apical 4-chamber view just above the tips of the tricuspid and mitral valves. **(See Figure 6, page 12.)** Generally, a ratio >1 is pathologic. In 2 retrospective studies (n=161 and n=1416), progressively higher values had higher specificity for pathology.[35,36] A major pitfall of the RV/LV ratio is its lack of effectiveness in biventricular disease, since both ventricles

Figure 5. Right Ventricular Myocardial Infarction on Right-Sided Electrocardiogram



V_{4-6} are V_4R-V_6R. Elevations in the inferior distribution (II, III, aVF) are associated with subtle elevation in V_4R concerning for right ventricular myocardial infarction.

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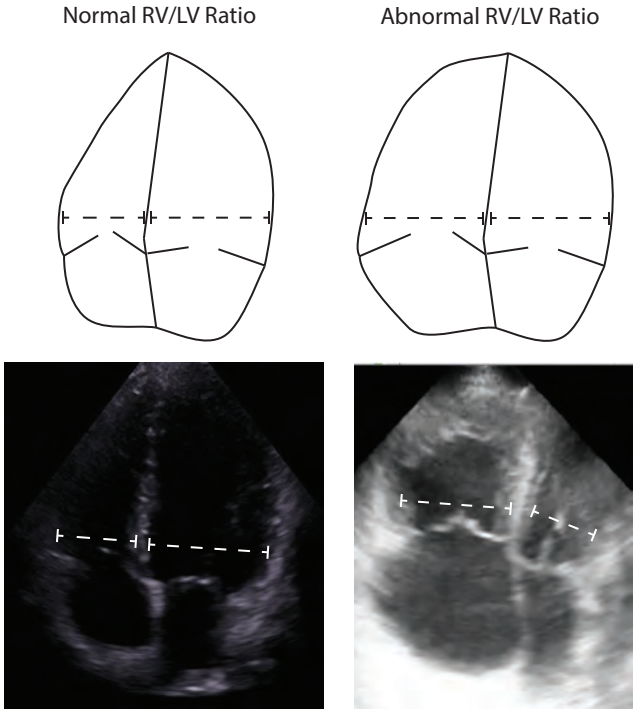
may be enlarged proportionally. In biventricular disease, measuring right ventricular end-diastolic diameter may be more effective, with measurements >4.2 cm representing elevated right ventricular pressure.

TAPSE measures the vertical movement of the lateral annulus of the tricuspid valve across the cardiac cycle. As much as 80% of right ventricular contraction occurs in the apical-basal plane. In a meta-analysis of 81 studies, greater vertical movement of the valve annulus was correlated with greater right ventricular systolic function.[37] To measure TAPSE, an apical 4-chamber view is obtained with M-mode crossing the lateral tricuspid annulus, and measurement is performed at the peak and trough of the waveform. **(See Figure 7.)** TAPSE values <17 mm are concerning for significant RVD and, in multiple prospective observational trials, were associated with worse outcomes in conditions including pulmonary embolism and acute heart failure.[22,31,38,39]

While TAPSE is a generally robust measure of overall right ventricular systolic function, it can be falsely normal in patients with severe chronic pulmonary hypertension. As the right ventricle remodels in chronic pulmonary hypertension, a greater proportion of right ventricular stroke volume comes from contraction in the lateral-septal plane as opposed to the typical apical-basal plane. Thus, when the right ventricle is chronically dilated due to long-standing severe pulmonary hypertension, TAPSE may be falsely preserved (ie, normal) in the presence of RHF.

Figure 6. Right Ventricle/Left Ventricle Ratio on Apical Echocardiography



Abnormal RV/LV suggests right heart pressure overload.
Abbreviation: RV/LV, right ventricle/left ventricle size ratio.
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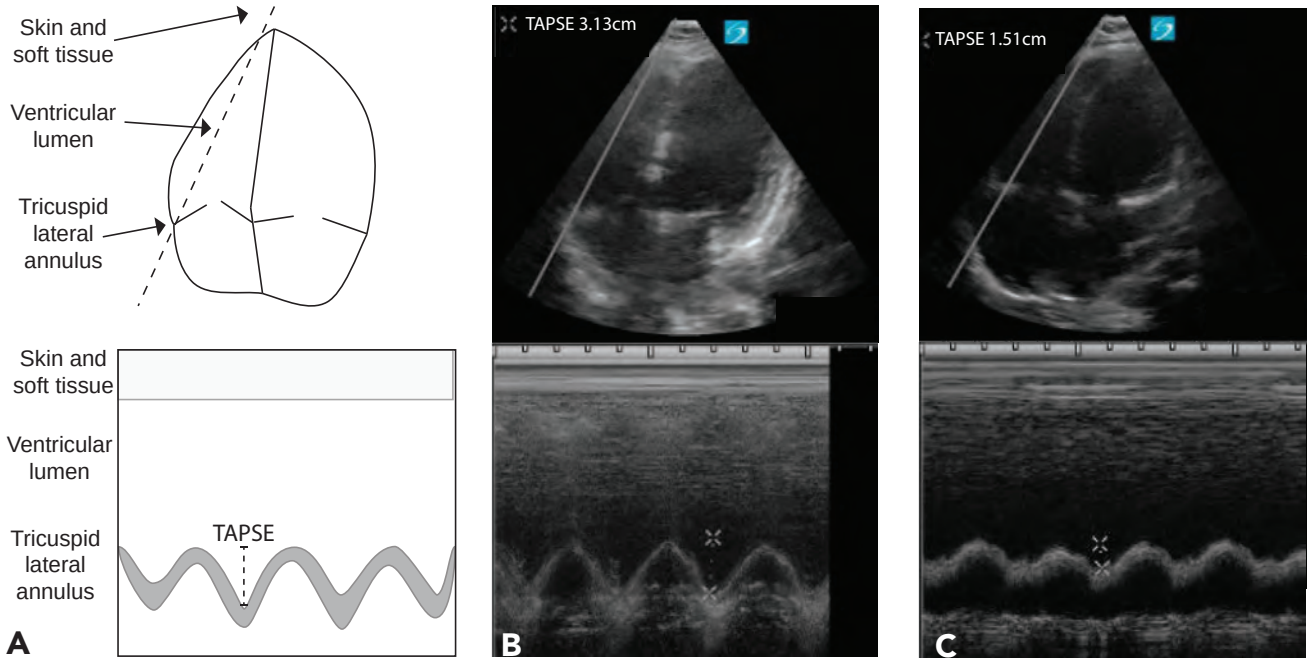
Figure 7. Tricuspid Annular Plane Systolic Excursion



Tricuspid annular plane systolic excursion (TAPSE) is the apical-basal movement of the lateral tricuspid annulus (Image A), typically measured by M-mode (dotted line) in an apical view. TAPSE ≥17 mm is a reliable cutoff for normal (Image B) versus abnormal (Image C) right ventricular systolic function.
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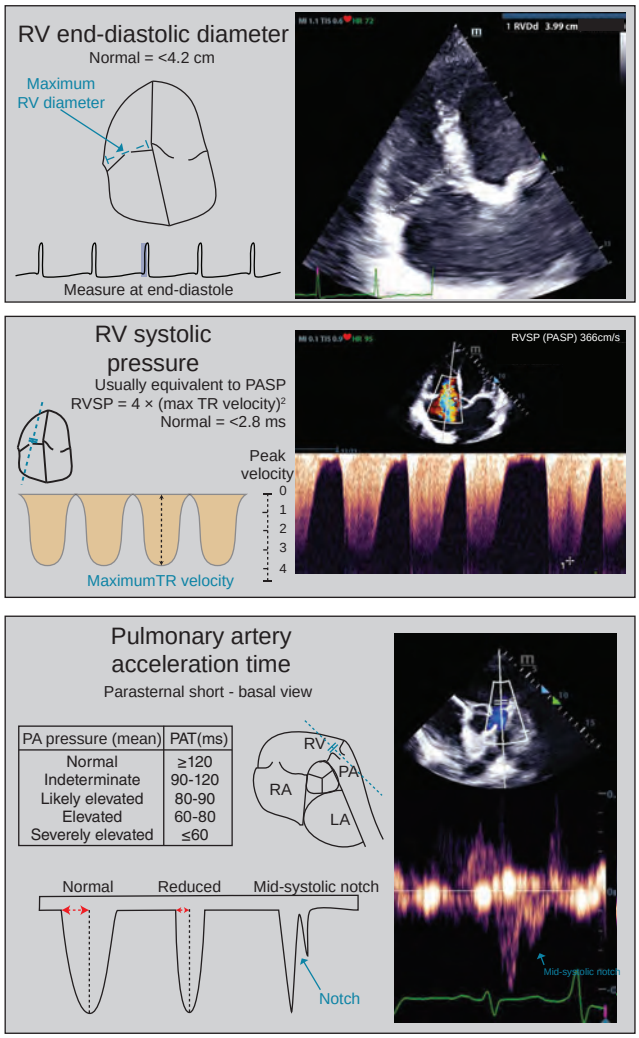
Prospective trials of 120 and 381 patients demonstrated that indexing TAPSE to the TTE-determined right ventricular systolic pressure or the right ventricular basal end-diastolic diameter may improve predictive value in patients with pre-existing severe chronic pulmonary hypertension.[38,40]

Beyond RV/LV ratio and TAPSE, other findings can help assess for RVD in specific situations.[41] Speckle tracking to estimate right ventricular strain has demonstrated promise as a more sensitive and specific measure of right ventricular function, but it is a relatively advanced technique that is not yet widely practiced in the ED.[22,38]

Figure 8. Abnormalities in Right Ventricular Pressure



Abnormalities in right ventricular end-diastolic diameter, right ventricular systolic pressure, and pulmonary artery acceleration time can help in diagnosis of right heart failure.

Abbreviations: PA, pulmonary artery; PAT, pulmonary acceleration time; PAAT, pulmonary artery acceleration time; RV, right ventricular; RVSP, right ventricular systolic pressure; TR, tricuspid.

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Right ventricular end-diastolic diameter >4.2 cm suggests elevated right ventricular pressures.[41] **(See Figure 8.)** Right ventricular systolic pressure (RVSP) which, in most circumstances, equals pulmonary artery systolic pressure (PASP), is estimated from the maximum velocity of the tricuspid regurgitant jet (present in approximately 90% of the healthy population)[1] on continuous-wave Doppler. Peak tricuspid regurgitant jet velocity >2.8 meters/sec is abnormal, and RVSP (PASP) is equal to 4 times this velocity squared.[41] **(See Figure 8.)** Many ultrasound machines facilitate automatic calculation of RVSP. Pulsed-wave Doppler of the pulmonic valve/right ventricular outflow tract can also suggest elevated pulmonary artery pressures. A mid-systolic notch in this waveform or a time to peak acceleration of <90 milliseconds is highly specific for elevated mean pulmonary artery pressure. Tracing the right ventricle chamber areas at end-systole and end-diastole in an apical view and calculating the percent difference yields right ventricular fractional area change, which is an alternative measure of right ventricular systolic function, with an abnormal cutoff at <35%.[41]

Right atrial pressure is estimated by inferior vena cava maximum diameter and collapsibility, just proximal to the junction of the hepatic vein and inferior vena cava.[41] **(See Figure 9, page 14.)** Two cutoffs are relevant: maximum inferior vena cava diameter ≥2.1 cm, and inferior vena cava respiratory collapsibility of ≤50%. If both are true, right atrial pressure is elevated (≥15 mm Hg, range 10-20 mm Hg); if both are false, right atrial pressure is normal (3 mm Hg, range 0-5 mm Hg).[41] Otherwise, right atrial pressure is indeterminate (8 mm Hg; range, 5-10 mm Hg). Compared with invasive measures, this estimation is highly accurate at values that are low (3 mm Hg) and high (15 mm Hg), but less so when indeterminate (8 mm Hg), or in the presence of invasive positive pressure ventilation.[41]

Computed Tomography

CT is essential in diagnosis of acute pulmonary embolism and may secondarily suggest the presence of RHF. RV/LV ratio >0.9 is the most-used CT marker of RHF. In moderately sized prospective studies of ED patients with pulmonary embolism, CT-derived RV/LV ratios are moderately sensitive (88%-93%) but poorly specific (39%-79%) for TTE determination of RVD,[42] with a 49% positive predictive value and an 83% negative predictive value.[43] Interestingly, in 862 patients undergoing CTPA, the prevalence of an RV/LV ratio >0.9 was similar in those with versus without acute pulmonary embolism.[44] This suggests that not only is an elevated RV/LV ratio common on CT, but also may reflect a patient's underlying comorbidities rather than the pulmonary embolism.[44]

Other CT findings may suggest RVD but have varying utility and/or feasibility. Retrospective data suggest that cardiac chamber volume on CT corre-

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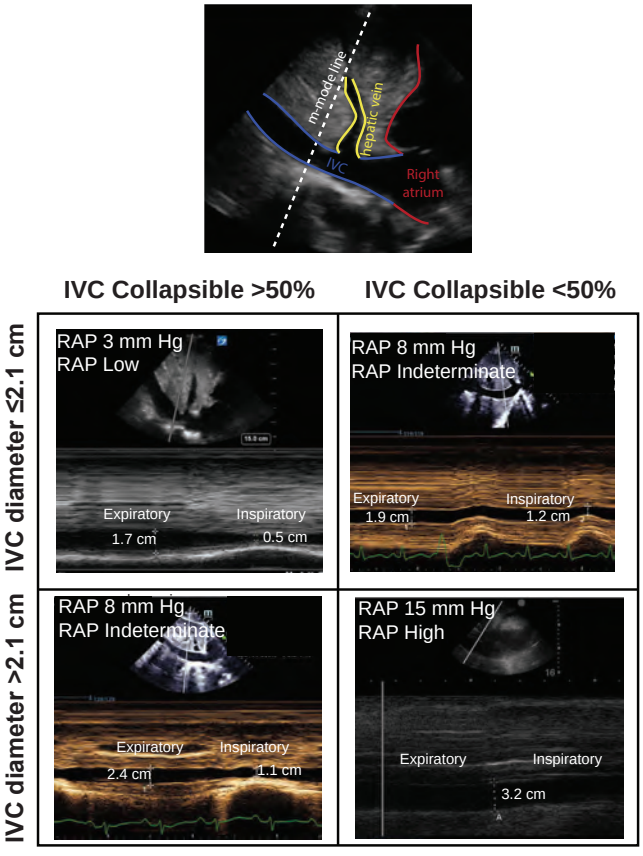
lates with myocardial injury in PE[45] and has greater area under the curve for diagnosing RVD than CT-derived RV/LV ratio (0.93 vs 0.84),[46] but is rarely measured in radiology reports. In a retrospective study of 47 patients, clot burden on CTPA correlated only modestly with both CT and TTE findings of RVD,[47] while location (central vs peripheral) correlated more strongly in a retrospective study of 252 patients.[6] A prospective study of 85 patients suggested that inferior vena cava reflux of contrast and bowing of the interventricular septum may also suggest RVD, although their significance is less well-studied.[48] Multiple large prospective studies demonstrated that ED patients with persistent dyspnea and a negative CTPA (no acute pulmonary embolism, pulmonary pathology, or other abnormal findings) had a 30% to 53% rate of isolated RVD, defined as TTE evidence of pulmonary hypertension or right ventricular hypokinesis without left ventricular dysfunction.[28,49,50]

Figure 9. Right Atrial Pressure Estimation by Ultrasound Measurement of Inferior Vena Cava Maximum Diameter and Collapsibility



Abbreviations: IVC, inferior vena cava; RAP, right atrial pressure.
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Magnetic Resonance Imaging

Cardiac magnetic resonance imaging can be useful in outpatient or inpatient evaluations to assess for infiltrative disease, myocarditis, and other pathology, but it is not recommended for ED evaluation of patients with RHF.

Summary of Diagnosis of Right Heart Failure

Figure 10, page 15 summarizes the ED evaluation of RHF. Once diagnosis is established, signs suggesting decompensated RHF include: worsening or severe acute heart failure symptoms, hemodynamic instability, worsening TTE and biomarkers (natriuretic peptides, troponin) compared to prior values, presence of a new etiology of RHF, and/or disproportionate impairment of TTE measures of right ventricular systolic function (TAPSE, fractional area change, free-wall strain) compared to measures of right heart pressure (RVSP, RV end-diastolic diameter, RV/LV ratio, right ventricular outflow tract acceleration time).[41] Right ventricular systolic function that is relatively preserved compared to right heart pressure may suggest chronic RHF that is more appropriately compensated. Right atrial pressure can also help in determining compensation. Nevertheless, caution should be exercised; low right atrial pressure can suggest compensated right ventricular function, but could be a sign of hypovolemia instead.

Treatment

Treatment of acute RHF is complex and varies based on the underlying etiology. For all etiologies of acute RHF, society guidelines emphasize the importance of treating triggering factors, managing volume status, optimizing oxygenation, aggressively treating hypotension with early vasopressor therapy, and considering advanced therapies for patients with refractory symptoms.[4] For patients with pulmonary arterial hypertension, it is crucial to avoid interruption or discontinuation of the patient's home pulmonary vasodilators to prevent hemodynamic collapse. In addition to this general approach to treatment, it is important to identify the cause or causes of acute right heart failure and apply etiology-specific treatment.

Revascularization

In cases of RVMI with acute RHF, treatment is largely similar to that of acute MI, with revascularization as the definitive therapy. Unique challenges include increased risk for bradyarrhythmias, ventricular tachycardia, and septal rupture.[5] Treatments indicated for stabilization include atropine for low-grade atrioventricular block or hemodynamically significant sinus bradycardia, pacing for third-degree block, and synchronized cardioversion for tachydysrhythmias.[5] While nitrates have historically been contraindicated in RVMI due to a rationale of preload dependence,

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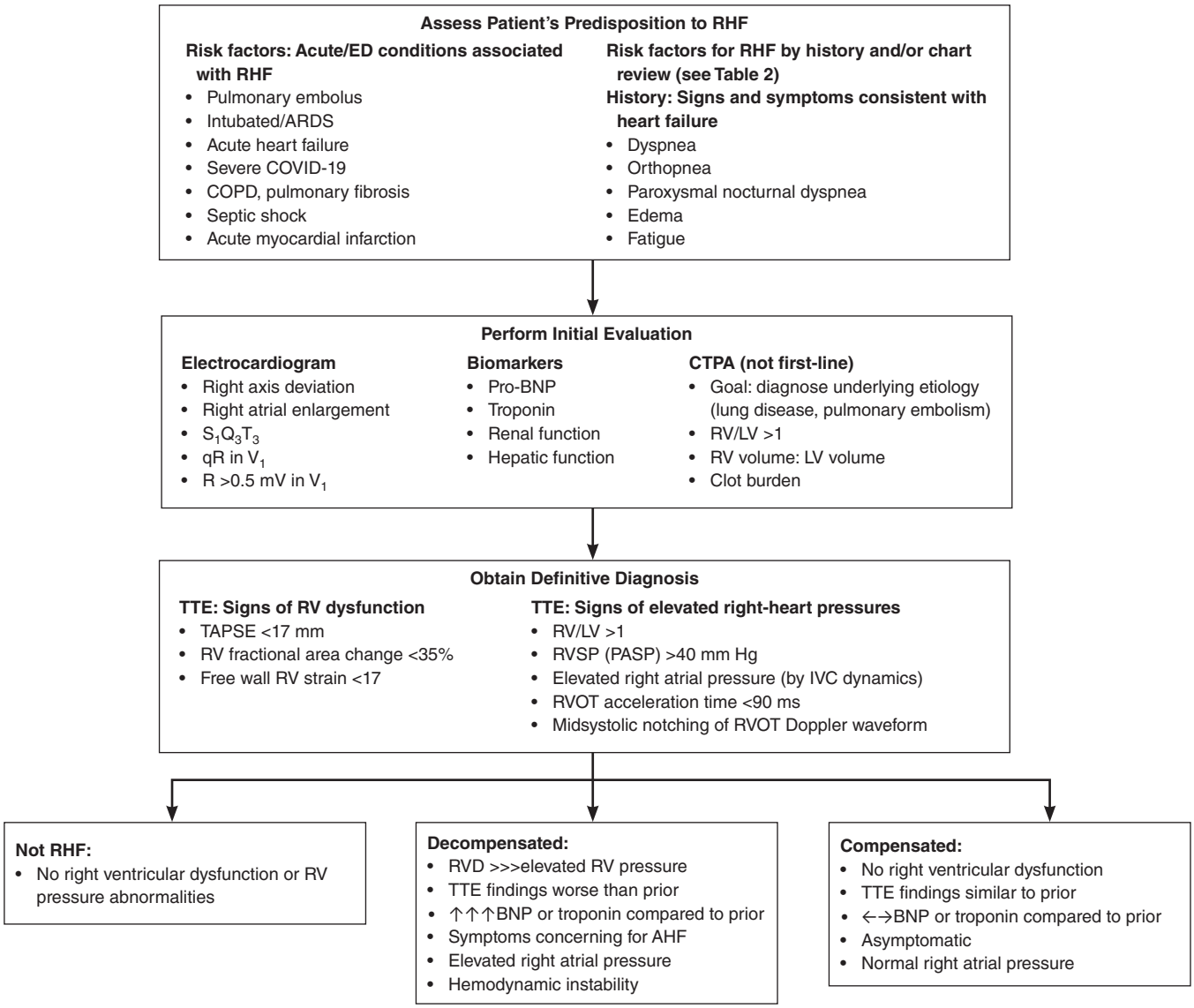
a 2023 meta-analysis with 1050 patients found no significant association with adverse events.[51]

Optimization of Preload

Volume status should be assessed based on history, physical examination, echocardiographic findings, and underlying etiology. Right atrial pressure can be assessed by inferior vena cava ultrasound. The relationship between preload and RHF is complex, and the common dogma that "the right heart is preload-dependent" can oversimplify clinical reality.[5] In patients with signs of hypovolemia and/or low right atrial pressure, augmenting preload with small IV boluses of crystalloid fluid (250-500 mL at a time) may support hemodynamics until euvolemia is reached and right atrial pressure begins to rise. However, acute RHF is often caused or exacerbated by volume/pressure overload, since elevated right atrial pressure and central venous congestion confer much of the morbidity in RHF.[5]

For patients with RVD and evidence of hypervolemia and/or elevated right atrial pressure, society guidelines recommend that volume status be managed with 40 to 80 mg IV furosemide if the patient is loop-diuretic naïve.[3] Those with signs of acute heart failure who are taking oral diuretics at home should receive 1 to 2.5 times their home dose (mg-to-mg conversion) intravenously.[52] In 4 studies

Figure 10. Diagnostic Approach to Right Heart Failure in the Emergency Department



Abbreviations: AHF, acute heart failure; ARDS, acute respiratory distress syndrome; BNP, brain natriuretic peptide; BUN, blood urea nitrogen; COPD, chronic obstructive pulmonary disease; CTPA, computed tomographic pulmonary angiography; ED, emergency department; IVC, inferior vena cava; PASP, pulmonary artery systolic pressure; RV, right ventricle/ventricular; RV/LV: ratio of size of RV to LV; RVOT, right ventricular outflow tract; RVSP, right ventricular systolic pressure; TAPSE, tricuspid annular plane systolic excursion; TTE, transthoracic echocardiography.

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comparing diuretic therapy to volume expansion in submassive pulmonary embolism, including 2 randomized trials, one or more measures of clinical stability and hemodynamics (heart rate, blood pressure, oxygen support, some echocardiographic measures, biomarkers) improved with diuretic therapy.[53-56] The specific measure that improved was not always consistent between studies. In none of the 4 studies did any measure of hemodynamics or clinical stability improve with volume expansion. None were powered for patient-oriented outcomes, and larger studies are needed. Overall, these data suggest that volume expansion is not hemodynamically or clinically favorable in this population, while diuretic therapy appears safe and may confer uncertain hemodynamic benefit.[53-56]

For patients with RVMI, diuresis was associated with more favorable hemodynamic improvement than saline loading.[57] For patients with acute heart failure, intensive treatment with higher-dose diuretics and/or vasodilators improves right ventricular function during hospitalization,[22,58] and ED/in-hospital decongestion intensity (ie, change in a quantitative measure of congestion) strongly correlates with lower 30-day death or acute heart failure-related readmission outcomes.[59]

Respiratory Support

Treating hypoxia and limiting hypercapnia are critical during RHF management. Hypoxemic pulmonary vasoconstriction increases right ventricular afterload, while acidosis impairs right ventricular systolic function.[1] Hypoxia vasoconstricts pulmonary vascular beds and causes ventilation/perfusion mismatch, while hypoventilation impairs carbon dioxide clearance and thereby causes or worsens acidosis. Society guidelines recommend that oxygenation should initially be optimized via passive oxygen therapy (nasal cannula, nonrebreather mask, or high-flow nasal cannula) to maintain oxygen saturation (SpO_2) >90%.[5]

Respiratory failure etiologies that are typically treated with noninvasive positive pressure ventilation (NIPPV) (eg, acute heart failure and acute chronic obstructive pulmonary disease [COPD] exacerbations) should utilize NIPPV unless hypotension develops. Few data exist for NIPPV versus passive oxygen delivery in the subset of patients with RHF and acute heart failure or acute COPD, but NIPPV use was associated with improved TAPSE in a 2022 prospective study of 84 patients.[22] NIPPV is associated with a relatively smaller increase in transpulmonary pressure compared with intubation. Ideally, optimization of oxygenation and ventilation with NIPPV may prevent the need for more invasive ventilation and decrease the risk for hemodynamic collapse. Unlike intubation, NIPPV can be stopped immediately if the patient's blood pressure begins to drop.

In contrast, intubation of patients with RHF should be avoided when possible. In a retrospective study of 340 patients, the risk for peri-intubation cardiac arrest and hemodynamic instability more than doubled with pre-existing moderate RVD (OR, 2.65) and severe RVD (OR, 2.66).[60] For patients who remain hypoxic despite maximal medical therapy, it is essential to optimize oxygenation status, acidosis, and hemodynamics prior to intubation to minimize the risk for decompensation.[5] If post-intubation hypotension does occur, it is essential to lower the PEEP and/or driving pressure to minimize the impact on right ventricular afterload, titrating to hemodynamic effect.[5] The maximum hemodynamically "safe" PEEP or plateau pressure will vary by patient and situation, so using the lowest possible pressures to achieve adequate oxygenation and ventilation is recommended, based on several small prospective trials.[14,61-63]

Vasopressors and Inotropes

Vasopressor and inotrope management in acute RHF is based predominantly on expert consensus and physiological theory.[5] **(See Figure 1, page 4 and Figure 2, page 5.)** A target mean arterial pressure of 65 mm Hg is reasonable, though this can be individualized based on response to therapy. Vasoactive agents should be limited to patients with current or impending shock.[5]

According to the American Heart Association and European Society of Cardiology, norepinephrine is the first-line vasopressor for hypotension and hypoperfusion.[5] In a prospective study of 27 patients, norepinephrine effectively raised systemic vascular resistance without significant deleterious effects on the pulmonary vasculature.[64] Epinephrine should be considered second-line or third-line therapy, due to the greater association with death and refractory shock compared to norepinephrine that was noted in a small randomized trial on post-MI cardiogenic shock.[65] Vasopressin is another second-line option for refractory shock, with theoretical benefit in RHF for reducing pulmonary vascular resistance, although this benefit has not been tested.[5] Phenylephrine should be avoided in patients with acute RHF, as it can cause vasoconstriction of the pulmonary vasculature.[5]

Inotropes may be beneficial for patients with severe ongoing right ventricular systolic dysfunction and congestion despite preload optimization and persistent clinical instability despite vasopressors. Dobutamine is a selective beta-1 adrenergic agonist, while milrinone is a phosphodiesterase (PDE3) inhibitor with theoretical effects for pulmonary vasodilation in addition to inotropy.[5] A randomized controlled trial of 192 patients did not demonstrate any benefit for milrinone versus dobutamine.[66] In a meta-analysis of 359 patients, levosimendan improved contractility without increasing myocardial oxygen demand via calcium sensitization, with favorable hemodynamic effects for RHF.[67] Levosimendan is approved in many countries, but not the United States. All inotropes cause systemic vasodilation, potentially worsening

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hypotension, and should therefore be initiated after vasopressors.[5]

Mechanical Circulatory Support

Based on expert consensus, the use of short-term mechanical circulatory support such as temporary right ventricular assist device or venoarterial extracorporeal membrane oxygenation (ECMO), is indicated for patients with RHF and cardiogenic shock refractory to medical management.[68] Mechanical circulatory support offers the possibility of bridging patients until recovery. The most important factor for patient selection is early initiation, prior to the development of multiorgan failure, after which prognosis is dismal.[4] Thus, early consideration of transfer to a mechanical circulatory support-capable specialty center is essential for critically ill acute RHF patients. Delaying transfer until after medical therapy fails risks severe multiorgan failure and missing the narrow window during which initiating mechanical circulatory support is most likely to be beneficial.

Special Populations

Patients on Pulmonary Vasodilators

Continuous prostacyclin infusions for home care of patients with pulmonary arterial hypertension are noteworthy because pump malfunction is a true emergency. Withdrawal from prostacyclin therapy may cause fulminant escalation in pulmonary vascular resistance, precipitating acute-on-chronic RHF and hemodynamic collapse. Pump malfunction should therefore be managed by immediately restarting the patient's continuous therapy and rapid consultation with their pulmonary arterial hypertension specialist. Oral phosphodiesterase 5 (PDE5) inhibitors and endothelin receptor antagonists are also used in the chronic management of pulmonary arterial hypertension, for which similar withdrawal/discontinuation effects are possible.[1]

Inhaled nitric oxide is a pulmonary vasodilator used in acute RHF.[4] In a small retrospective study, inhaled nitric oxide was found to have positive hemodynamic effects on patients with acute RHF, including reduced pulmonary vascular resistance, mean pulmonary artery pressure and central venous pressure, as well as increased cardiac output.[69] Patients with submassive pulmonary embolism showed improved hemodynamics in a randomized placebo-controlled trial of nitric oxide, but patient-oriented outcomes were neutral.[70]

Controversies and Cutting Edge

Speckle-Tracking Echocardiography

Right ventricle speckle-tracking echocardiography (STE) is increasingly used by cardiologists. In a prospective observational trial of 62 patients, STE improved right ventricular assessments, compared to conventional TTE measures.[58] While some data using STE in ED patients to assess RVD exist,[38] feasibility is limited by ED clinician training, uptake, and access to STE-enabled platforms. Although cardiac magnetic resonance imaging, 3D-echocardiography, and right-heart catheterization are the gold standards in the evaluation of RHF, none of them are readily available or feasible for emergency clinician use at the present time, and TTE is considered the noninvasive reference standard for diagnosing and grading RHF in the acute care setting.[1,5,41]

Advanced Therapies

Treatment for patients with submassive pulmonary embolism (ie, patients with RVD who are normotensive) remains highly controversial. In an 83-patient randomized trial that was terminated prematurely, systemic thrombolysis had significantly lower rates of a composite adverse outcome than anticoagulation alone.[71] However, results were primarily driven by improved functional capacity and recurrent pulmonary embolism at 30 days, rather than death, shock, or intubation. Comparison of 13,240 patients from a nationwide registry receiving systemic thrombolysis, anticoagulation, or catheter-directed therapies failed to show a mortality benefit for systemic thrombolysis or catheter-directed therapies versus anticoagulation (adjusted OR, 0.61; 95% CI, 0.34-1.1), though the analysis was not adjusted for blood pressure.[72]

Decisions to initiate advanced therapies (systemic thrombolysis, catheter-directed therapies, thrombectomy) should be made at the local level with specialist consultation or after transfer to a center with such expertise. Implementation of standardized protocols for the evaluation and risk stratification and management of pulmonary embolism may help expedite and optimize care.[73]

Similarly controversial is the role for empiric systemic thrombolysis in cardiac arrest presumed to be due to acute pulmonary embolism with acute RHF, for which few data exist. This is a challenging clinical scenario when the pulmonary embolism diagnosis has not been previously confirmed, since TTE is relatively inaccurate for making etiology-specific diagnosis and CTPA is not possible during cardiac arrest. Furthermore, it is speculated that perfusion during cardiopulmonary resuscitation may be inadequate for IV systemic thrombolysis to be effective. Nevertheless, in the absence of rapidly available mechanical circulatory support or thrombectomy, systemic thrombolysis therapy is reasonable if there is high concern for acute pulmonary embolism in cardiac arrest, given an absence of better options.

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Case Conclusions

CASE 1

For the 40-year-old woman with dyspnea, tachycardia, and hemoptysis...

After CT confirmation of a diagnosis of pulmonary embolism, you performed a bedside TTE and diagnosed RVD based on an increased RV/LV ratio. You recognized the need for hospital admission due to her high risk for hemodynamic collapse, and you consulted your hospital pulmonary embolism response team for the consideration of advanced therapies.

CASE 2

For the 64-year-old man with heart failure who was experiencing severe fatigue and dyspnea...

You performed a bedside TTE and identified biventricular failure, based on a TAPSE of 13 mm and inferior vena cava dynamics of elevated right atrial pressure. Because this conveys a high risk for major adverse events, you admitted him to the hospital after administering double his home diuretic dose.

CASE 3

For the 52-year-old woman with COVID-19 in severe respiratory distress following intubation...

You recognized significant RVD, with a TAPSE of 11 mm on bedside TTE. Thinking quickly, you reduced PEEP and tidal volume and tolerated mild hypoxia, and the patient's vasopressor requirements immediately decreased dramatically. You admitted her to the ICU, alerting the ICU team that mechanical circulatory support may be necessary if her hemodynamic instability persisted.

Disposition

Patients with incidentally discovered chronic pulmonary hypertension and no obvious acute process are commonly identified in the ED, particularly after negative CTPA for dyspnea.[28,49,50] Assuming clinical stability, these patients are typically appropriate for ED discharge with referral to a pulmonary hypertension specialist for outpatient follow-up, since etiologies such as CTEPH and pulmonary arterial hypertension can be reversible with etiology-specific treatments guided by a pulmonary hypertension specialist.

5 Things That Will Change Your Practice

1. Utilize TTE to identify RHF/RVD/pulmonary hypertension in at-risk populations, and to inform disposition.
2. The RV/LV ratio on CTPA may not add value to risk stratification of patients with pulmonary embolism.
3. In patients with RHF, minimize invasive PPV (reduce PEEP and/or tidal volume and avoid intubation, if possible). Use pressors early.
4. Negative CTPA with dyspnea often indicates undiagnosed pulmonary hypertension in ED patients.
5. Volume resuscitation is indicated to treat only hypovolemia. In decompensated RHF, excess preload is more often a problem than low preload.

Patients With Pulmonary Embolism

Virtually all patients with pulmonary embolism and RHF will require hospital admission due to the high risk for morbidity and mortality, even if they would otherwise be identified as low-risk by algorithms such as the simplified pulmonary embolism severity index (sPESI).[73] Based on multiple recent meta-analyses, in patients with hemodynamically stable acute pulmonary embolism, TTE-determined RVD (with measures including TAPSE <17 mm and enlarged RV/LV ratio) is associated with reduced survival, risk for impending hemodynamic collapse, and other adverse events.[75-77] Employing a strategy to identify RVD in otherwise low-risk pulmonary embolism patients improves the identification of patients at risk for short-term adverse events compared to risk-stratifying acute pulmonary embolism by other clinical features.[74,78] Whether an abnormal RV/LV ratio on CT is predictive of mortality in low to intermediate risk acute pulmonary embolism is more controversial, with recent meta-analyses finding opposing conclusions.[8,75-77,79] Elevations in troponin and natriuretic peptides are associated with adverse event risk in patients with acute pulmonary embolism,[8] although whether they add incremental predictive value to TTE is similarly controversial.[8,76]

Patients With Acute Decompensated Heart Failure

For patients with acute decompensated heart failure, the presence of RVD is a powerful predictor of adverse events overall and in comparison to the left ventricular ejection fraction.[2,21,22,38,40,58,80-82] In a recent ED-based study, TAPSE <17 mm and other right heart measures were stronger predictors of adverse

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events than left heart measures, and they added incremental value for identifying low-risk patients able to be discharged, compared to a validated clinical decision instrument.[22,38] History of COPD, lower left ventricular ejection fraction, greater pulmonary edema severity (B-lines) on lung ultrasound, and greater right ventricular systolic pressure on TTE are independently associated with RVD in acute heart failure patients.[22]

Patients With Congenital Heart Disease

Adult patients with a history of congenital heart disease presenting for decompensated RHF require careful consideration in disposition. In a single observational study, patients who had successful repair of their congenital disease had similar outcomes as general adult patients with acute heart failure.[23] Nevertheless, such patients typically follow up with a subspecialist in adult congenital heart disease, and it may be reasonable to contact their subspecialist for recommendations. In contrast, children with congenital heart disease will almost always need subspecialty consultation and transfer to a pediatric specialty hospital, since their congenital heart disease is often either unrepaired or newly repaired and may require acute surgical intervention.

Patients Requiring Higher Levels of Care

The presence of RHF is a poor prognostic factor in many clinical contexts. Patients with RHF in the context of severe COVID-19, ARDS, and/or acute cor pulmonale are at a dramatically increased risk for adverse events compared to those without RHF.[13,15-17,60,62,83] Given a 21% prevalence of RHF in the ARDS population, identifying RHF to triage these patients to a higher level of care within the hospital or via transfer to a specialty center is essential. The dramatic difference in mortality and other serious outcomes suggests that patients with RHF should be triaged to a higher level of care in an intensive care unit if not already intubated or potentially transferred to a specialty center with ECMO capabilities.

A similar approach is necessary with regard to patients with sepsis and acute MI complicated by RHF.[9,11,84-87] In both cases, patients with RVD have substantially worse outcomes and are more likely to experience early decompensation after the ED course (death, heart failure, cardiogenic shock).[9,11,84-87] Accordingly, patients with sepsis or acute RVMI with acute RHF should be triaged to a higher level of care or transferred to a specialty center.

Patients presenting to the ED with decompensated pulmonary arterial hypertension and RHF should be admitted to the hospital. Consultation with the patient's pulmonary arterial hypertension specialist should occur in the ED, since such patients often receive complex specialty medications and because transfer to a specialty center may be indicated. Pulmonary arterial hypertension patients with tachypnea, signs of infection, and renal dysfunction are at particularly high risk and should be triaged to higher level of care.[88,89]

Summary

RHF is a complex condition caused by and/or complicating diverse etiologies including pulmonary embolism, pulmonary arterial hypertension, MI, acute heart failure, congenital heart disease, pulmonary disease, ARDS, and more. Historical factors, signs and symptoms of heart failure, ECG, and natriuretic peptide and troponin levels are key in identifying patients at risk for RHF, while TTE is critical for confirmation and risk stratification.

Decompensated RHF is challenging to manage. Keys include early assessment of volume status and right atrial pressure, offloading excess preload when right atrial pressure is elevated, avoiding hypoxia and acidosis, supporting hemodynamics, and reversing the underlying cause with etiology-specific therapy. Some treatment goals have inherent tradeoffs with one another (eg, intubation vs treatment of hypoxia, treating hypovolemia with crystalloid vs avoiding volume overload), and must be balanced carefully to prevent cardiovascular collapse and death. Relatively simple indices such as RV/LV ratio, TAPSE, and inferior vena cava ultrasound estimation of right atrial pressure can offer useful diagnostic and prognostic information. When shock persists despite aggressive support with vasopressors and inotropes, early transfer to a specialty center able to initiate mechanical circulatory support is typically necessary. Regardless of etiology, decompensated RHF portends elevated risk for death and other adverse events, making admission to a higher level of care and early consideration of consultation or transfer critical for disposition.

Time- and Cost-Effective Strategies

- Bedside TTE is an effective tool in assessing volume status and guiding diuresis or fluid resuscitation needs in patients with RHF.
- Emergency physicians are accurate at acquiring and interpreting many TTE signs of RHF on a bedside examination, with minimal training, compared to formal TTE.
- TAPSE is easy and accurate for emergency clinicians to learn on bedside ultrasound.
- The most important element in diagnosing RHF is considering comorbidities that can cause RHF.
- Patients with refractory RHF should be transferred to a tertiary care facility for advanced therapeutics.

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Risk Management Pitfalls for Emergency Department Patients with Right Heart Failure

1. **"I ruled out pulmonary embolism, so the right ventricle dilation was meaningless."** Right ventricle dilation on a negative CTPA is associated with conditions such as pulmonary arterial hypertension that should be referred to a specialist.

2. **"They weren't hypotensive from their pulmonary embolism, so TTE wasn't helpful."** RVD is a major predictor of adverse events in normotensive patients with acute pulmonary embolism.

3. **"I did not obtain TTE before discharge because their sPESI score showed them to be at low risk."** RVD on TTE predicts adverse events in pulmonary embolism even when clinical scores show low risk.

4. **"I ruled out pulmonary embolism with the normal TTE."** TTE confirms the presence or absence of RHF, but does not indicate the underlying etiology.

5. **"I ruled out pulmonary embolism before I intubated them. Why did they arrest?"** RHF is common to several etiologies beyond pulmonary embolism, and is associated with greater risk of peri-intubation arrest.

6. **"They had pulmonary edema on their chest x-ray, how could they have had RHF?"** Acute heart failure is biventricular in up to half of cases.

7. **"I used push-dose phenylephrine during intubation, which should have prevented their arrest."** Phenylephrine can exacerbate RHF/pulmonary hypertension, and is relatively contraindicated in these patients.

8. **"I kept bolusing fluids because pulmonary hypertension is 'preload dependent.' I don't know why they crashed."** The classic dogma that patients with RHF are "preload-dependent" is an oversimplification. It is more common in decompensated RHF that right atrial pressure is high, for which fluids may worsen RVD.

9. **"I withheld diuresis for their acute RHF because of their acute kidney injury."** Patients with acute RHF usually develop renal dysfunction as a consequence of elevated right atrial pressure, and not low cardiac output.

10. **"There's nothing that transfer to a tertiary hospital could have done that my local hospital couldn't do in order to prevent them arresting."** Treatments for decompensated RHF requiring a specialty center's expertise—particularly mechanical circulatory support and advanced pulmonary embolism therapies—are most beneficial when initiated rapidly before multiorgan failure develops.

Class of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.
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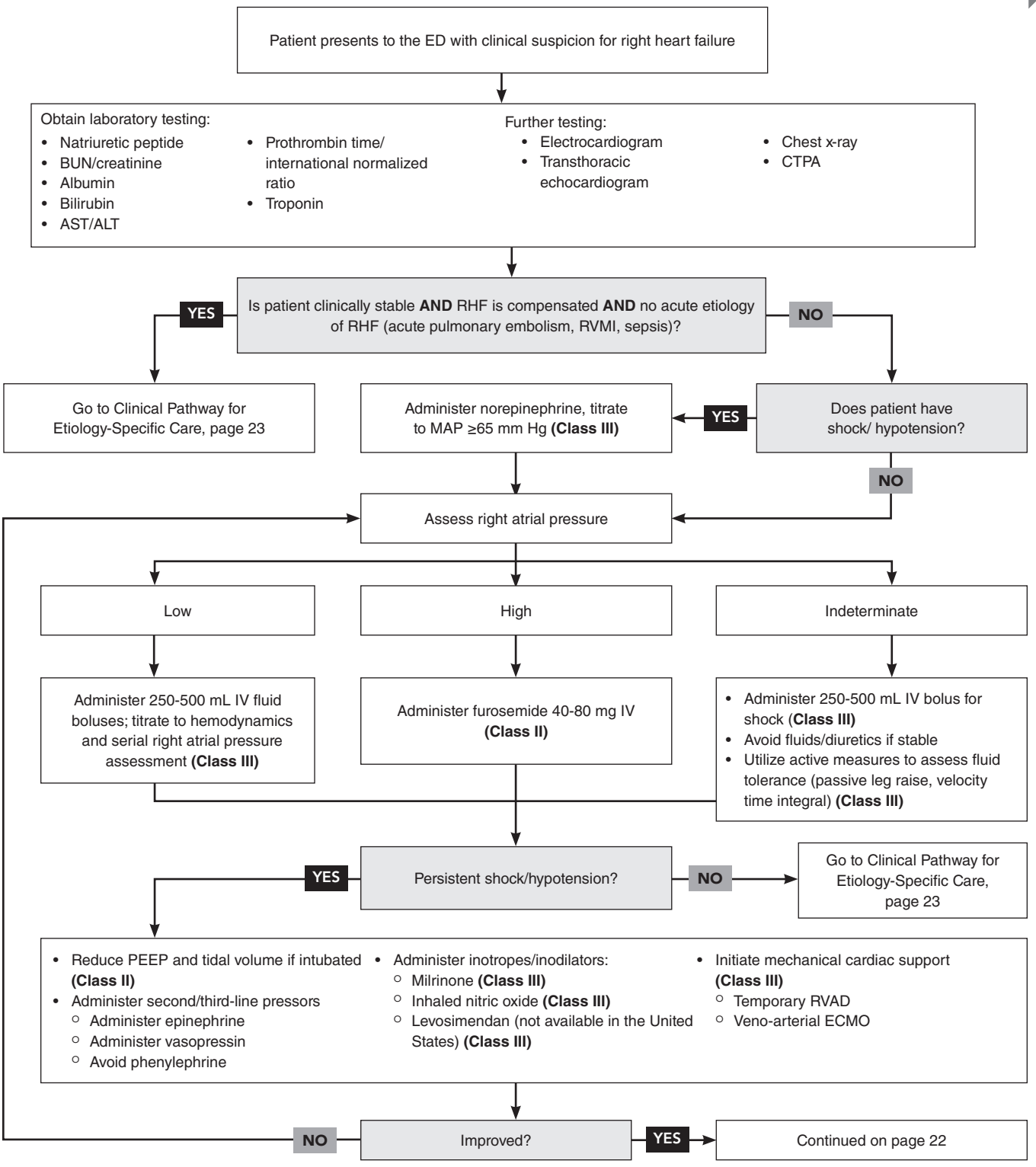
Clinical Pathway for Clinical Suspicion for Right Heart Failure in the Emergency Department


Patient presents to the ED with clinical suspicion for right heart failure
Obtain laboratory testing:
Natriuretic peptide
BUN/creatinine
Albumin
Bilirubin
AST/ALT
Prothrombin time/ international normalized ratio
Troponin
Further testing:
Electrocardiogram
Transthoracic echocardiogram
Chest x-ray
CTPA
Is patient clinically stable AND RHF is compensated AND no acute etiology of RHF (acute pulmonary embolism, RVMI, sepsis)?
YES
NO
Go to Clinical Pathway for Etiology-Specific Care, page 23
Administer norepinephrine, titrate to MAP ≥65 mm Hg (Class III)
YES
Does patient have shock/ hypotension?
NO
Assess right atrial pressure
Low
High
Indeterminate
Administer 250-500 mL IV fluid boluses; titrate to hemodynamics and serial right atrial pressure assessment (Class III)
Administer furosemide 40-80 mg IV (Class II)
Administer 250-500 mL IV bolus for shock (Class III)
Avoid fluids/diuretics if stable
Utilize active measures to assess fluid tolerance (passive leg raise, velocity time integral) (Class III)
YES
Persistent shock/hypotension?
NO
Go to Clinical Pathway for Etiology-Specific Care, page 23
Reduce PEEP and tidal volume if intubated (Class II)
Administer second/third-line pressors
Administer epinephrine
Administer vasopressin
Avoid phenylephrine
Administer inotropes/inodilators:
Milrinone (Class III)
Inhaled nitric oxide (Class III)
Levosimendan (not available in the United States) (Class III)
Initiate mechanical cardiac support (Class III)
Temporary RVAD
Veno-arterial ECMO
NO
Improved?
YES
Continued on page 22

Abbreviations: ALT, alanine transaminase; AST, aspartate transaminase; BUN, blood urea nitrogen; CTPA, computed tomographic pulmonary angiogram; ECMO, extracorporeal membrane oxygenation; ED, emergency department; INR, international normalized ratio; IV, intravenous; MAP, mean arterial pressure; PEEP, positive end-expiratory pressure; PT, prothrombin time; RHF, right heart failure; RVAD, right ventricular assist device; RVMI, right ventricular myocardial infarction.

For Class of Evidence definitions, see page 20.

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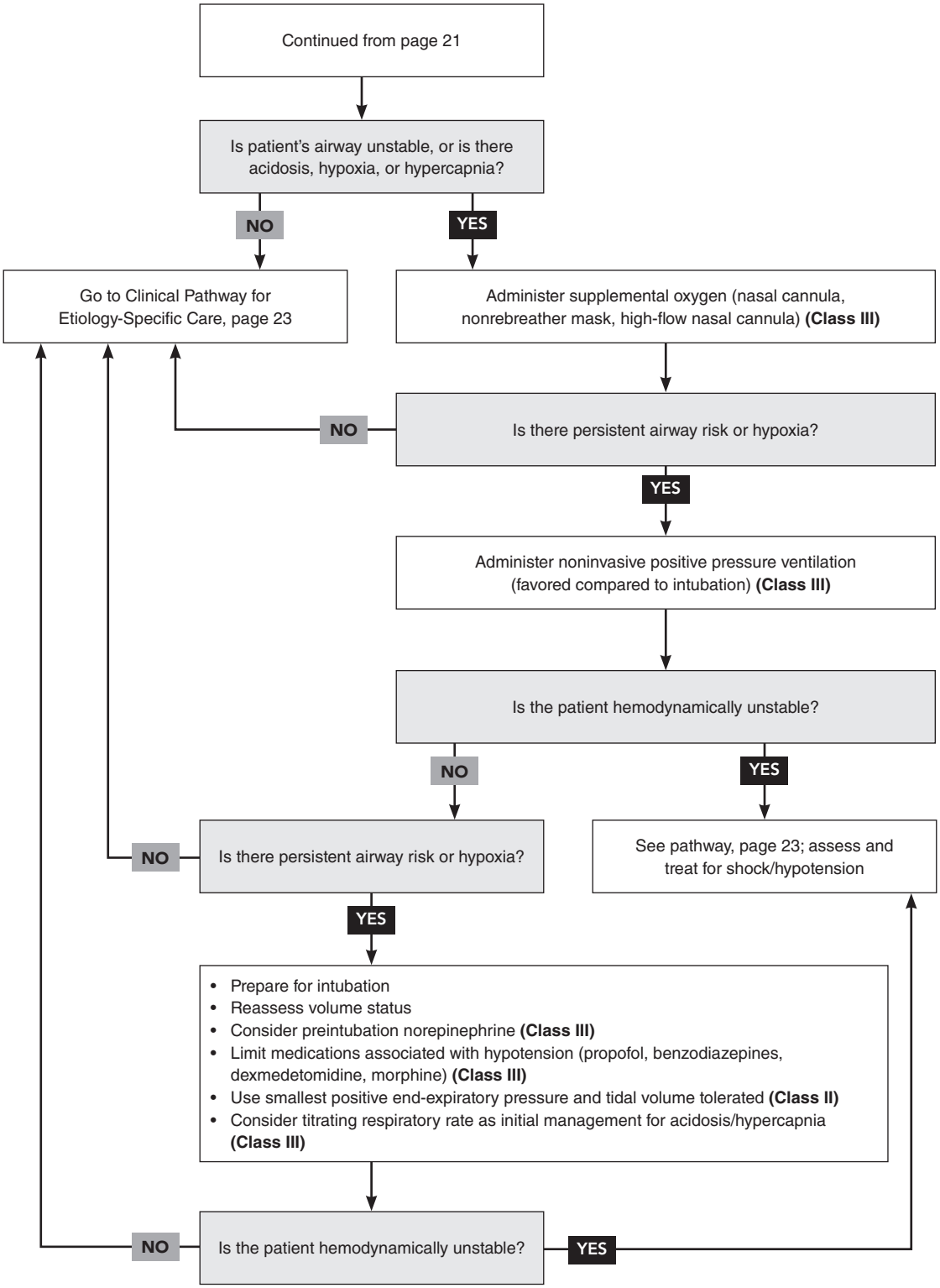
Clinical Pathway for Clinical Suspicion for Right Heart Failure in the Emergency Department (Continued)


Continued from page 21
Is patient's airway unstable, or is there acidosis, hypoxia, or hypercapnia?
NO
YES
Go to Clinical Pathway for Etiology-Specific Care, page 23
Administer supplemental oxygen (nasal cannula, nonrebreather mask, high-flow nasal cannula) (Class III)
NO
Is there persistent airway risk or hypoxia?
YES
Administer noninvasive positive pressure ventilation (favored compared to intubation) (Class III)
Is the patient hemodynamically unstable?
NO
YES
NO
Is there persistent airway risk or hypoxia?
See pathway, page 23; assess and treat for shock/hypotension
YES
• Prepare for intubation
• Reassess volume status
• Consider preintubation norepinephrine (Class III)
• Limit medications associated with hypotension (propofol, benzodiazepines, dexmedetomidine, morphine) (Class III)
• Use smallest positive end-expiratory pressure and tidal volume tolerated (Class II)
• Consider titrating respiratory rate as initial management for acidosis/hypercapnia (Class III)
NO
Is the patient hemodynamically unstable?
YES

For Class of Evidence definitions, see page 20.

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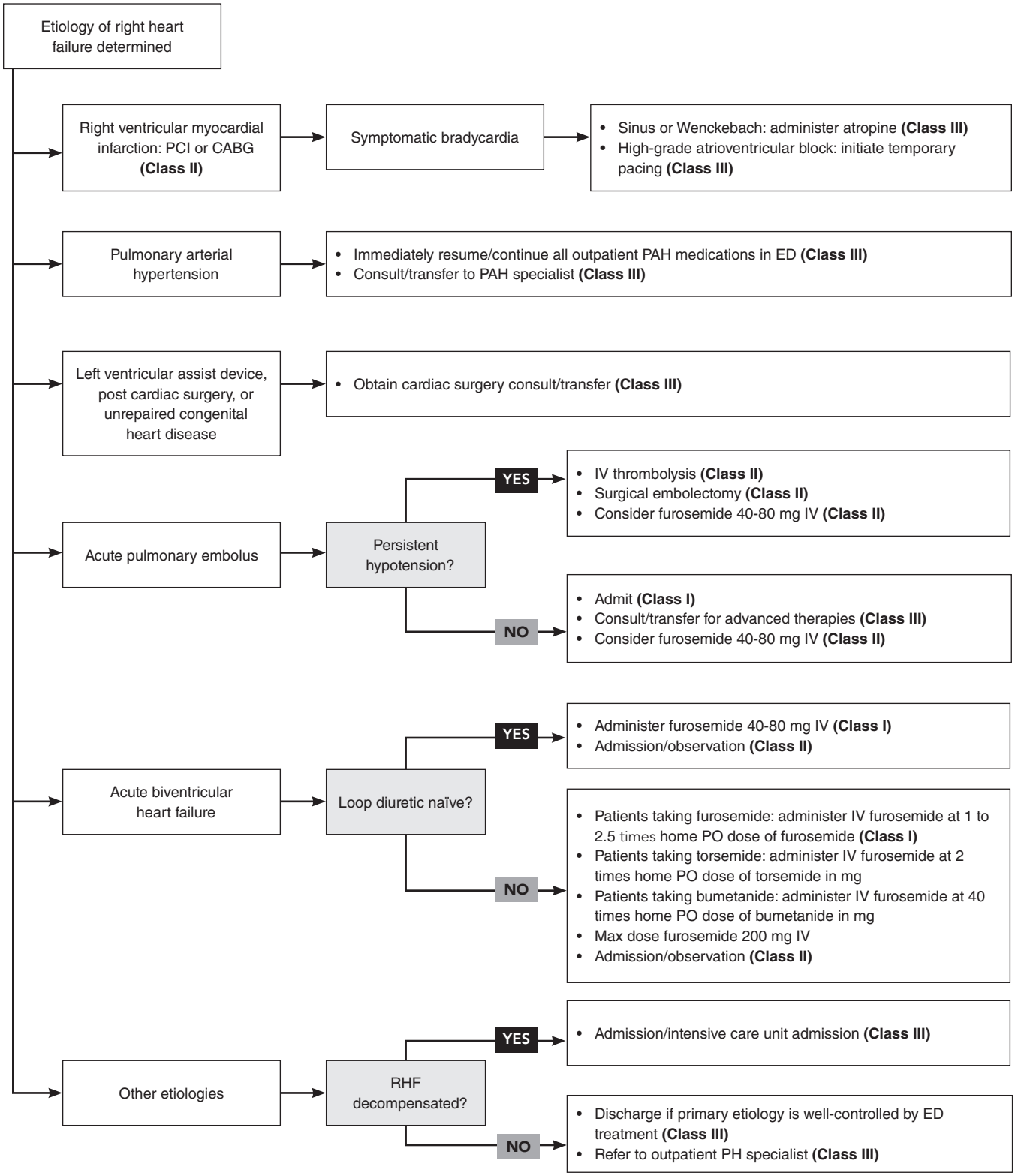
Clinical Pathway for Etiology-Specific Care of Right Heart Failure in the Emergency Department


Etiology of right heart failure determined
Right ventricular myocardial infarction: PCI or CABG (Class II)
Symptomatic bradycardia
• Sinus or Wenckebach: administer atropine (Class III)
• High-grade atrioventricular block: initiate temporary pacing (Class III)
Pulmonary arterial hypertension
• Immediately resume/continue all outpatient PAH medications in ED (Class III)
• Consult/transfer to PAH specialist (Class III)
Left ventricular assist device, post cardiac surgery, or unrepaired congenital heart disease
• Obtain cardiac surgery consult/transfer (Class III)
Acute pulmonary embolus
Persistent hypotension?
YES
• IV thrombolysis (Class II)
• Surgical embolectomy (Class II)
• Consider furosemide 40-80 mg IV (Class II)
NO
• Admit (Class I)
• Consult/transfer for advanced therapies (Class III)
• Consider furosemide 40-80 mg IV (Class II)
Acute biventricular heart failure
Loop diuretic naïve?
YES
• Administer furosemide 40-80 mg IV (Class I)
• Admission/observation (Class II)
NO
• Patients taking furosemide: administer IV furosemide at 1 to 2.5 times home PO dose of furosemide (Class I)
• Patients taking torsemide: administer IV furosemide at 2 times home PO dose of torsemide in mg
• Patients taking bumetanide: administer IV furosemide at 40 times home PO dose of bumetanide in mg
• Max dose furosemide 200 mg IV
• Admission/observation (Class II)
Other etiologies
RHF decompensated?
YES
• Admission/intensive care unit admission (Class III)
NO
• Discharge if primary etiology is well-controlled by ED treatment (Class III)
• Refer to outpatient PH specialist (Class III)

Abbreviations: CABG, coronary artery bypass grafting; ED, emergency department; IV, intravenous; PAH, pulmonary arterial hypertension; PCI, percutaneous coronary intervention; PH, pulmonary hypertension; PO, oral; RHF, right heart failure.

For Class of Evidence definitions, see page 20

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References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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(Randomized controlled trial; 276 patients)
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1. **Which ventilator change should be considered first if there is concern for acute cor pulmonale?**
   a. Decrease respiratory rate
   b. Increase tidal volume
   c. Reduce positive end-expiratory pressure (PEEP)
   d. Decrease fraction of inspired oxygen (FiO_2)

2. **Approximately what proportion of acute heart failure involves right heart dysfunction?**
   a. 5%
   b. 10%
   c. 20%
   d. 50%

3. **Which biomarkers of hepatic dysfunction are more strongly associated with shock rather than congestion in right heart failure (RHF)?**
   a. Bilirubin
   b. Alkaline phosphatase
   c. Alanine transaminase (ALT) and aspartate transaminase (AST)
   d. Albumin

4. **What is the tricuspid annular plane systolic excursion (TAPSE) cutoff that suggests a high risk for pulmonary embolism and acute heart failure?**
   a. <17 mm
   b. <21 mm
   c. <25 mm
   d. <29 mm

5. **What is the right atrial pressure interpretation of an inferior vena cava diameter of 2.5 cm, with 48% respiratory collapse?**
   a. 3 mm Hg, normal
   b. 5 mm Hg, low-normal
   c. 8 mm Hg, indeterminate
   d. 15 mm Hg, high

6. **In normotensive patients with acute heart failure, what is the primary driver of renal dysfunction?**
   a. Low cardiac output
   b. Elevated central pressure/volume overload (or venous congestion)
   c. Arterial vasoconstriction
   d. Over-diuresis/pre-renal

7. **For a patient with acute pulmonary embolism with right ventricular dysfunction, elevated right atrial pressure (≥15 mm Hg) by inferior vena cava ultrasound, and initially normal blood pressure, which would be the preferred approach to preload management?**
   a. 250-mL bolus of crystalloid fluid
   b. 500-mL bolus of crystalloid fluid
   c. 1000-mL bolus of crystalloid fluid
   d. 40 to 80 mg IV furosemide

8. **Which of the following approaches to hypoxia is LEAST LIKELY to cause hemodynamic compromise in RHF?**
   a. Noninvasive ventilation
   b. Intubation with ARDS-Net (acute respiratory distress syndrome) settings
   c. Intubation with propofol
   d. Awake intubation

9. **Which vasopressor should be avoided in patients with pulmonary hypertension/RHF?**
   a. Norepinephrine
   b. Phenylephrine
   c. Vasopressin
   d. Epinephrine

10. **What is the most appropriate ED management of a patient with pulmonary arterial hypertension who presents because their prostacyclin pump is malfunctioning?**
    a. Arrange follow-up and discharge to get a new pump as an outpatient.
    b. Immediately start their prostacyclin as IV, consult their pulmonary arterial hypertension specialist.
    c. Immediately start milrinone and admit to intensive care unit.
    d. Transfer to a referral center where the prostacyclin can be restarted on arrival.

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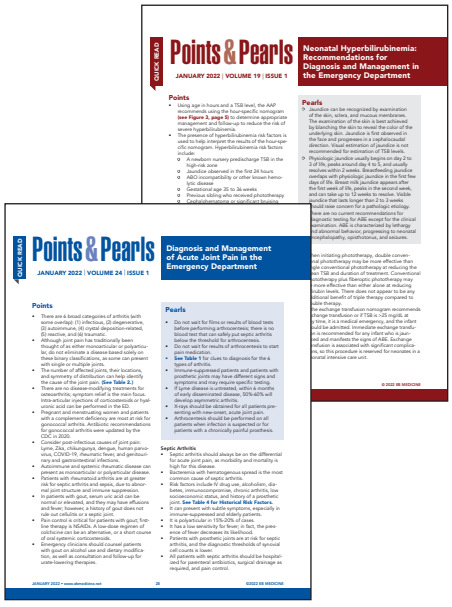
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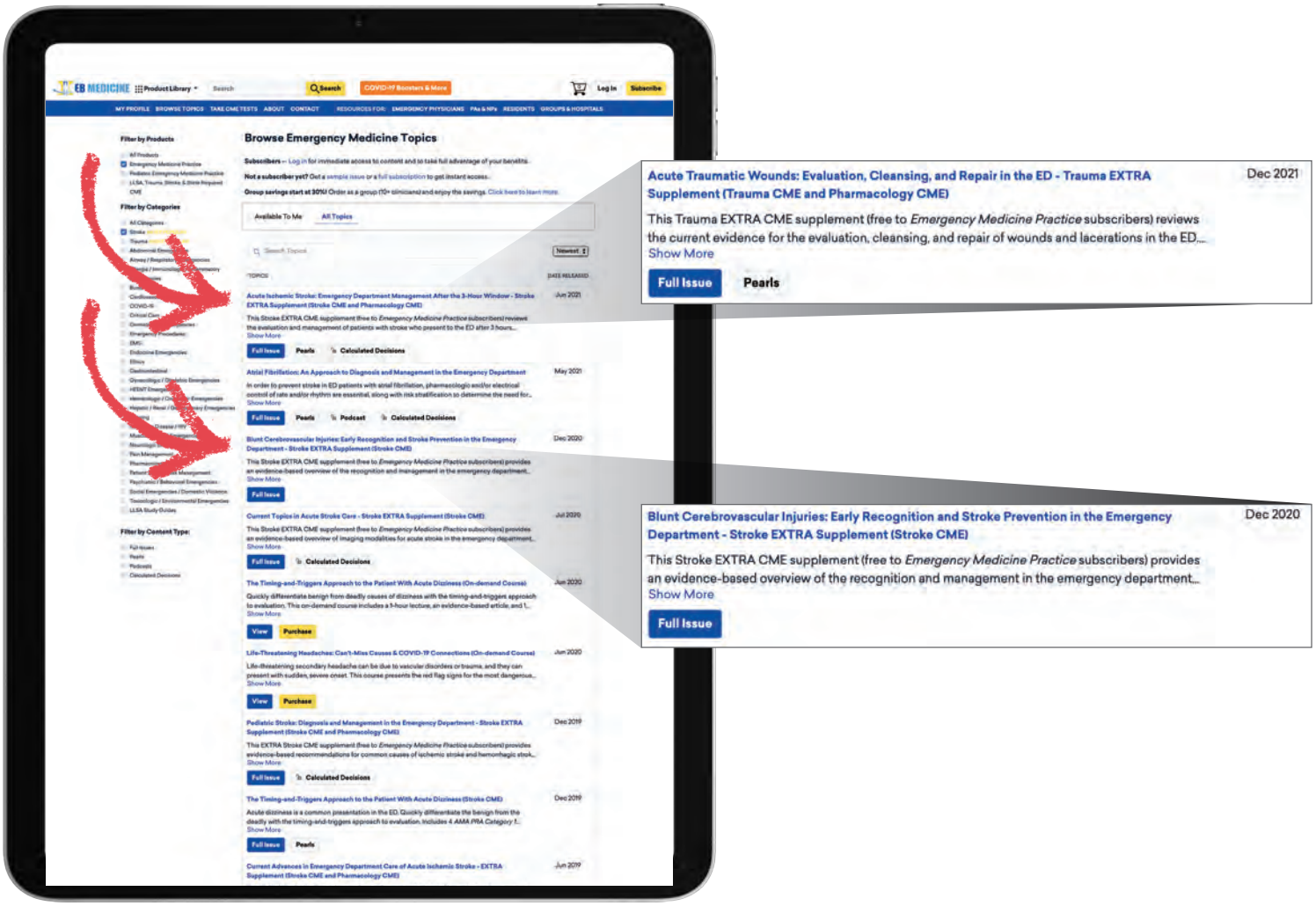
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QUICK READ

Points & Pearls

FEBRUARY 2024 | VOLUME 26 | ISSUE 2

Emergency Department Management of Patients With Right Heart Failure

Points

- Right heart failure (RHF) is the common endpoint of numerous cardiac, pulmonary, and systemic pathologies.
- Recognition of RHF in the ED is vitally important, as it can dramatically alter the management of commonly presenting conditions.
- Right heart function is dictated by 3 physiologic domains: (1) resistance to right ventricular ejection (afterload) via the pulmonary artery; (2) right ventricular systolic and diastolic pump function; and (3) right heart filling (preload) via systemic venous return. **(See Figure 1.)**
- The possible causes of RHF are vast, and more than one acute or chronic etiology may contribute. **See Table 2** for etiologies of acute and acute-on-chronic RHF. **Table 4** lists common ED diagnoses associated with acute RHF.
- RHF has nonspecific symptoms similar to left-sided heart failure, including fatigue, exercise intolerance, shortness of breath, chest pain, lower extremity edema, syncope, early satiety, and arrhythmia.[3,4]
- Causes of acute RHF include acute pulmonary embolism, sepsis, right ventricular myocardial infarction, positive pressure ventilation, acute respiratory distress syndrome, and COVID-19.
- Causes of chronic RHF include chronic thromboembolic pulmonary hypertension, heart failure, congenital heart disease, left ventricular assist devices, lung disease, and pulmonary arterial hypertension.
- ED evaluation for RHF should include a thorough history and chart review for risk factors. **(See Table 1.)**
- No symptoms are highly sensitive or specific for RHF.[1,27]
- No physical examination signs are sensitive or specific for RHF. Auscultation lacks significant accuracy for diagnosing pulmonary edema.[1,22]
- Laboratory studies are useful in screening for etiologies of RHF, but they lack specificity for confirming the diagnosis.
- A 12-lead electrocardiogram may aid in screening for RHF; signs are summarized in **Table 5**.

Pearls

- Pulmonary hypertension is associated with nearly all etiologies of RHF as either a progressive chronic disease or by rapid severe decompensation. **See Table 1** for the classifications, etiologies, and comorbidities.
- Venous congestion is both a cause and effect of elevated right ventricular pressures and can become a self-propagating cycle. **(See Figure 2.)**
- Pulmonary embolism is the most common cause of acute RHF.
- Transthoracic echocardiography is the primary diagnostic modality for confirming the presence or absence of RHF, but it does not typically identify underlying etiologies.[5,31]
- A major pitfall of RV/LV ratio is its lack of effectiveness in biventricular disease, which is present in nearly half of heart failure patients.
- **Figure 10** outlines the diagnostic approach to RHF in the ED.
- Treatment of RHF is outlined in the **Clinical Pathways**.
- Treating hypoxia and limiting hypercapnia are critical in RHF management.
- Intubation should be avoided when possible.
- The ratio of the sizes of the right and left ventricle (RV/LV) and tricuspid annular plane systolic excursion (TAPSE) can provide assessment of RVD.
- **Figures 6, 7, 8, and 9** graphically demonstrate TTE measurement in diagnosis of RHF.
- CT is essential in diagnosis of acute pulmonary embolism and may suggest the presence of RHF.
- Disposition of patients with RHF is dependent on the etiology.
- Withdrawal from prostacyclin therapy may precipitate hemodynamic collapse; pump malfunctions should be managed immediately.
- Decision to initiate advanced therapies (systemic thrombolysis, catheter-directed therapies, thrombectomy) should be made with specialist consultation or transfer to a center with this expertise.

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